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BETA IRRADIATION IN OPHTHALMOLOGY

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THE MAJORITY of reports on radiation therapy of the eyes are concerned with gamma rays. The purpose of this paper, however, is to discuss the use of the less well known beta rays in the treatment of nonmalignant lesions of the lids and the anterior ocular segment.

Beta rays possess the same qualities as other rays in the power of selective tissue destruction, but, in contrast to gamma rays, they penetrate about 3 mm. of tissue only. This makes them ideally suited to treatment of the anterior ocular segment. In no single case in which this therapy has been used has any damage to the lens been observed. This has been true clinically, as well as in animal experiments, in which in 1 instance a rabbit cornea was given 300 gram seconds of beta rays without a resultant cataract.[1]

The ease of application is a great advantage in the use of the beta rays. In the removal of certain small benign tumors of the lids or the conjunctiva, the absence of pain, the fact that hospitalization is not required and the excellent cosmetic result all make beta radiation therapy peculiarly acceptable both to children and to adults, especially persons who object to surgical procedures. In the treatment of tuberculosis of the anterior ocular segment a certain analgesic effect has been noted and the course of the disease seems to be shortened. The symptoms of vernal conjunctivitis are decreased and the follicles disappear with treatment. Superficial vessels of the cornea can be occluded and new vascularization inhibited; thus, beta irradiation is ideal as postoperative therapy in cases of lamellar keratectomy, in preventing postoperative granulation and in inhibiting the overgrowth of connective tissue.

TREATMENT

Beta radiation is applied with a special radon applicator (fig. 1) designed by Mr. Curtis F. Burnam.[2] This consists of a 5 mm. soda glass bulb containing from

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.

1. Hughes, W. F., and Iliff, C. E.: The Effects of Beta Irradiation on the Rabbit Eye, Am. J. Roentgenol. **56**:502 (Oct.) 1946.

2. Burnam, C. F., and Neill, W., Jr.: Use of Beta Ray of Radium Applicator: Description of Method and Results Obtained in Superficial Lesions of Eye, South. M. J. **33**:279, 1940.

200 to 500 millicuries of radon, enclosed in a brass cylinder with walls 2 mm. thick. From a 4 mm. window at one end of the applicator, beta and gamma rays pass unfiltered, the alpha rays being stopped by the soda glass container.

The dose of beta radiation is calculated in gram seconds (the product of the weight of radium in grams, or its equivalent in curies of radon, and the exposure time in seconds). Since the skin erythema dose of unfiltered beta rays is 18 gram seconds, whereas that of gamma rays is 8 gram minutes, it is possible to give adequate beta radiation therapy with such short exposures that the effect of the gamma rays is negligible.

The applicator is placed as close to the lesion as possible (approximately 1 to 3 mm.) and moved slowly back and forth across an estimated square centimeter of field, giving a spray action to the rays. The distance of application is important, as the strength of the delivered dose varies inversely with the square of the distance from the source. Care should be taken that the overlapping effect does not exceed the calculated optimum dose. In the treatment of small tumors, pterygiums, granulation tissue or blood vessels invading the cornea, the applicator is held in contact with the lesion.

Beta irradiation has a cumulative effect if repeated doses are given within a period of two weeks. The total dose to any one area should not exceed 18 gram seconds during this period. With adults not more than 12 gram seconds is given to an area at one treatment; with children the dose is started at 3 to 4 gram seconds.

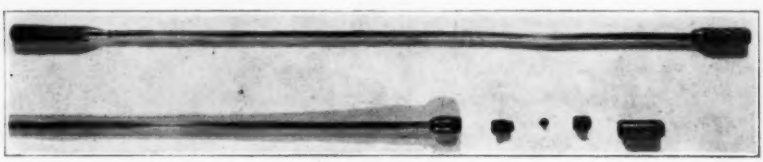

Fig. 1.—Burnam beta ray applicator (radon).

Before a maximum dose is administered, the individual sensitivity to beta rays should always be tested. The course of treatment consists of 18 gram seconds in a two week period, followed by a rest period of two to three weeks. At the end of this time another course of 18 gram seconds may be given. When the condition is not acute, the patient may be placed on a monthly maintenance dose of 8 to 10 gram seconds.

When contact therapy is used, or when photophobia, pain and lacrimation make adequate exposure impossible, local anesthesia with tetracaine hydrochloride is helpful. The treatments themselves cause no pain, but an exacerbation of the existing symptoms frequently occurs in three to four hours and may last twenty-four hours. In such cases a mild sedative is helpful.

In some instances a punctate stippling of the cornea (clearly visible when stained with fluorescein) has appeared within twenty-four hours and lasted four to five days. Loss of cilia is rare but does occur in heavily treated areas of the lids.

VERNAL CONJUNCTIVITIS

Throughout the literature on vernal conjunctivitis there are many reports on the excellent results with radium therapy. However, owing to scarcity of data and to variations in technic, evaluation of this material is difficult. An adequate review of the literature to 1929 was

given by Kumer and von Sallmann,[3] and since that time outstanding reports have been made by Quick,[4] Stallard,[5] Robinson,[6] Bowing and Fricke [7] and Pendergrass and Andrews.[8]

Material.—Data on 60 patients with vernal conjunctivitis who were treated with beta rays appear in table 1. All the patients were followed for at least one full season of nine months and some for as long as six years. The majority of white patients had exhibited symptoms before the age of 30; the majority of Negro patients, before the age of 10 years.

The total dose producing a cure ranged from 7 gram seconds, in an early lesion, to 258 gram seconds, spread over three and one-half years, in a case of a long-standing palpebral lesion. Most patients (70 per cent) showed decided symptomatic improvement with less than 40 gram seconds of beta radiation given as spray therapy.

A case illustration of the course of the disease and the rate of clearing with therapy is that of W. C., a 5 year old Negro boy, first seen in July 1941. Typical symptoms of lacrimation, photophobia, itching and discharge had been present during the previous season, and at the time of examination the lids had a velvety appearance but no follicles. The limbal vegetations were present around the upper half of each cornea from 3 to 9 o'clock (fig. 2 *A*).

Two treatments, with a total dose of 10 gram seconds, were given to the limbus, and four treatments, with a total dose of 20 gram seconds, to the upper lids. After these treatments he became free from symptoms and the vegetations flattened within three weeks (fig. 2 *B*). The following year there was a slight recurrence of the disease, and 9.4 gram seconds of beta radiation was given to the limbus and 7 gram seconds to the lids. The child had no further symptoms until the summer of 1943, when there was a mild recurrence, with lacrimation and itching. At this time the limbus of the left eye was treated with 9 gram seconds of radiation, and the right eye was used as a control. The left eye promptly cleared, whereas the right eye continued to give symptoms until it also received beta radiation therapy. For purposes of checking, the child was seen in the summer of 1944 and again in the spring of 1945; no recurrence of symptoms or signs was evident on either occasion.

The severest case of limbal vernal conjunctivitis seen in this series was that of C. O., a 7 year old Negro boy (fig. 3 *A*). The disease had been present four years, and vision was reduced to 10/200 in each eye. For three seasons the boy received therapy, the total dose being 80 gram seconds. Improvement was slow, but by the end of the first season there was definite gain, and by the end of the third season his vision had improved to 20/40 (fig. 3 *B*). He is still under treatment at the time of this report.

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3. Kumer, L., and von Sallmann, L.: Die Radiumbehandlung in der Augenheilkunde, Vienna, Julius Springer, 1929.

4. Quick, D.: Radium in Vernal Catarrh, Arch. Ophth. **4**:212 (Aug.) 1930.

5. Stallard, H. B.: Effect of Radium Emanations, Tr. Ophth. Soc. U. Kingdom **53**:224, 1933.

6. Robinson, G. A.: Benign Lesions of Eye, Ear, Nose and Throat, Am. J. Roentgenol. **33**:801, 1935.

7. Bowing, H. H., and Fricke, R.: Radium Treatment of Vernal Conjunctivitis, Am. J. Roentgenol. **38**:740, 1937.

8. Pendergrass, E. P., and Andrews, J. R.: The Radium Emanation Treatment of Vernal Catarrh, Am. J. Roentgenol. **34**:637, 1935.

TABLE 1.—*Data on Patients with Vernal Conjunctivitis*

Name	Race; Sex; Age, Yr.	Duration Before Treatment	Degree * and Area of Involvement		Total Dose, Gm. Sec.†	Time Under Observation	Result
			When First Seen	When Last Seen			
F. S.	W; M; 19	2½ years	Severe, 4 lids	Slight scarring, 3 lids	258 to upper; 16 to lower lids	6 years	Cure
C. W.	N; F; 5	Less than 1 yr.	Mild, upper lids and limbus	Clear	10.7, lids and limbus	1 year	Cure
W. S.	W; M; 42	2 years	Moderate, upper lids	Few follicles, upper lids	35	3 seasons	Improvement
M. G.	W; M; 29	1 year	Moderate, all 4 lids	Slight scarring, upper lids	1940: 29 (3) 1941: 36 (7) 1942: 36 (5); 101	4 years	Improvement
E. C.	W; F; 5	9 months	Moderate, all 4 lids	Still some injection of lids	16.8, lower lid; 12, upper lid	2 seasons	Improvement
E. K.	N; F; 18	Less than 1 yr.	Mild, limbus	Cure	7	3 years	Cure
W. C.	N; M; 5	Less than 1 yr.	Moderate, palpebral and limbal areas	Cure	1941: 10.9 to limbus, 20 to lids; 1942: 9.4 to limbus, 7.5 to lids; 1943: 18 to limbus of left eye	1941-1946 5 seasons	Cure
P. T.	N; F; 5	6 months	Mild, limbal area	Slight limbal elevation	6.55	2 seasons	Improvement
W. M.	W; M; 27	15 years	Moderate, 4 lids	Still some follicles, upper lids	46.4	2d season	No improvement
C. A.	N; M; 24	Unknown	Moderate, 4 lids and limbus	Slight scarring, upper lids	1939: 99 to lids, 76 to limbus; 1940: 19 to lids	3 years	Cure
S. D.	W; M; 42	3 years	Moderate, upper lids	Moderate, injection, upper lids	Heavily treated at Kelly ‡ and Wilmer	3 years	No improvement
P. C.	W; F; 17	1 year	Mild, upper lids	Slight injection, upper lids	30 (4)	1 season	Improvement
P.	W; F; 32	1 year	Moderate, upper lids	Cure	40 (5)	2 years	Cure
J. P.	W; M; 20	4 years	Moderate, 1 lid	Slight, 1 lid	32 (4)	1 season	Improvement
M. S.	W; F; 18	6 weeks	Moderate, 3 lids	Slight, 2 lids	16	1 season	Improvement
P.	W; F; 40	1 year	Moderate, upper lids	Slight, 1 lid	28	2 years	Improvement
R. R.	W; F; 40	6 years	Moderate, upper lids	Slight, upper lids	38 right eye only	2 seasons	Improvement
W. N.	W; M; 30	3 years	Moderate, upper lids	Slight, upper lids	18	1 season	Improvement
M. M.	W; M; 32	2 years	Moderate, upper lids	Slight, upper lids	30	1 season	Improvement
G. H.	W; M; 16	3 years	Moderate, 4 lids	Slight, upper lids	35	1 season	Improvement
S. H.	W; M; 30	2 years	Slight, upper lids	Slight, upper lids	18	1 year	Improvement
S. C.	W; M; 50	3 years	Slight, upper lids	Cure	18	1½ years	Cure
T. W.	W; M; 6	6 weeks	Slight, limbus	Cure	10	1 year	Cure
W. W.	W; M; 4	6 weeks	Slight, limbus	Cure	10	1 year	Cure
L. J.	N; M; 9	2 years	Moderate, upper lids	Moderate, upper lids	Left eye: 1943: 25 right eye: 1945: 10 left eye: 1946: 10	3 years	Improvement

M. W.	N; M; 6	2 years	Moderate, upper lids	Slight, 1 upper	88	2 seasons	Improvement
S. B.	W; M; 4	3 months	Mild, limbus	Cure	14	1 year	Cure
J. K.	W; M; 8	2 years	Moderate, upper lids	Mild, upper lids	24	1 season	Improvement
L. G.	W; F; 18	Several years	Upper lids and limbus	No limbal injection but upper lids still moderately affected	1941: 70.6 to lids; 1942: 56.2 to limbus	2 seasons	No improvement
A. M.	W; M; 12	1 year	Severe, upper lids	Occasional follicle	1942: 85.4 to right eye 19.6 to left eye	2 years	Improvement
V. G.	W; F; 10	3 years	Severe, upper lids	Some scarring, upper lids	Right eye: 108; left eye: 100	3½ years	Improvement
M. G.	W; M; 25	2 years	Moderate, upper lids	Mild, upper lids	21 to lids	2 seasons	Improvement
R. W.	N; F; 10	3 weeks	Mild, upper lids and limbus	Cure	15.9 to lids, 34 to limbus	2 years	Cure
H. M.	W; F; 5	8 months	Mild, upper lids and limbus	Cure	26.9 to lids, 15. 1 to limbus	2 seasons	Cure
M. C.	N; F; 7	2 years	Mild, upper lids	Cure	27.8	1 season	Improvement
E. P.	N; M; 10	1 year	Mild, limbus	Cure	20.2	2 seasons	Cure
W. M.	N; F; 6	3 years	Moderate, limbus	Eyes white	11.5	1 year	Improvement
L. Y.	W; M; 13	6 months	Mild, upper lids	Cure	18.4	3 years	Cure
C. O.	N; M; 9	4 years	Very severe, limbus	Moderate, limbus	80 to limbus, 25 to lids	5 seasons	Improvement
M. M.	W; M; 14	3 years	Severe, upper lids	Mild, upper lids	89.4 to lids (12)	2 years	Improvement
W. C.	W; M; 26	4 years	Severe, 4 lids	Cure	37.9 to upper and 11.4 to lower lids, 21 to 1 limbus	6 years	Cure
C. T.	N; M; 3	1 year	Moderate, limbus	Cure	23	1 season	Improvement
K. O.	W; M; 8	1 year	Severe, upper lids	Some scarring	82 (8)	2 years	Improvement
Y. W.	N; F; 5	3 weeks	Moderate, limbus	Slight, limbus right eye	17	1 season	Improvement
C. S.	W; F; 30	Unknown	Moderate, upper lids	Mild, upper lids	9	1 season	Improvement
T. J.	N; F; 16	1 year	Mild, lids and limbus	A few follicles, left upper lid	38 (5) to lids, 11.5 to limbus	2 seasons	Improvement
E. R.	N; M; 4	†	Mild, upper lids	No lesion	5	1 season	Improvement
B. B.	W; F; 3	1 year	Moderate, 4 lids	Mild, 4 lids	24 (3)	1 season	Improvement
D. J.	W; F; 9	3 weeks	Moderate, upper lids	No lesion	20	1 season	Improvement
V. R.	N; F; 4	6 months	Moderate, limbus	Mild, limbus, right eye	44 (5)	1 season	Improvement
D. F.	N; M; 2	1 week	Moderate, limbus	Mild, limbus	9	1 season	Improvement
W. S.	W; M; 12	1 year	Moderate, 4 lids	Cure	45 (5)	2 seasons	Cure
B. H.	W; F; 6	1 year	Moderate, 4 lids	Mild, upper lids	24 (2)	1 season	Improvement
J. W.	W; M; 24	6 months	Moderate, upper lids	Mild, right upper lid	38 (4)	1 season	Improvement
C. S.	W; F; 29	3 years	Moderate, 4 lids	Mild, 3 lids	50 (4)	1 year	Improvement
J. M.	W; F; 24	3 years	Moderate	.....	80 (8)	2 seasons	Improvement
M. C.	W; F; 31	3 years	Slight, upper lids	Cure	50 (4)	2 seasons	Cure
A. Z.	W; M; 5	1 year	Slight, upper lids	Cure	15 (2)	3 years	Cure
J. S.	W; M; 42	2 years	Slight, upper lids	Slight, upper lids	45	2 years	Improvement
S.	W; M; 25	1 year	Moderate, upper lids	Mild, upper lids	72 (6)	1 season	Improvement

* Severe involvement indicates the presence of large follicles, profuse discharge and prominent symptoms; moderate, the presence of some follicles, discharge and redness and moderately severe symptoms; mild, the presence of small or absent follicles, redness and/or slight discharge and few or no symptoms.

† Values in parentheses indicate number of treatments.

‡ Howard A. Kelly Hospital.

A case of a palpebral lesion is that of M. W., a 6 year old Negro boy. The symptoms had been present two years (fig. 3 *C*). He was given nine treatments at intervals of two to three weeks, the total dose equaling 88 gram seconds (fig. 3 *D*). The follicles disappeared, and he became almost free from symptoms. This child, if he can be reached, will be given two prophylactic treatments next season.

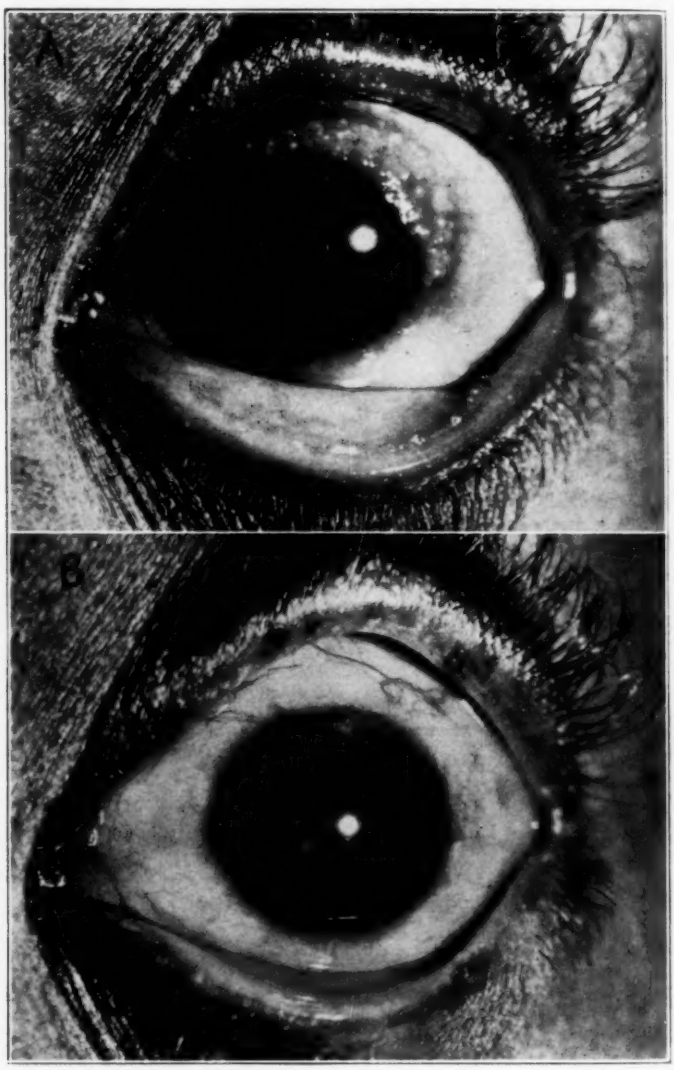

Fig. 2 (W. C.).—Limbus in a case of vernal conjunctivitis (*A*) before and (*B*) after treatment with 10 gram seconds of beta radiation.

Results.—The results of beta radiation therapy of 60 patients with vernal conjunctivitis are presented in table 2. Forty-one patients had

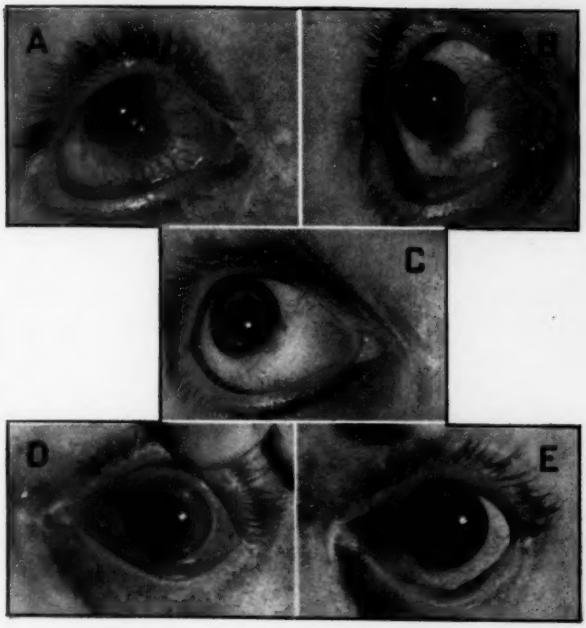

Figure 6

Figure 3

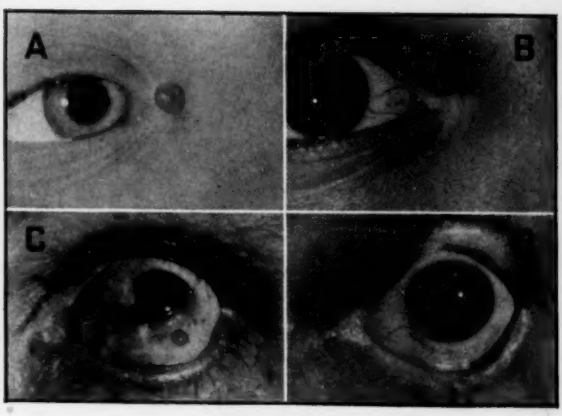

Figure 9

Fig. 3.—*A* (C. O.), severe limbal vernal conjunctivitis of four years' duration; *B*, same eye after treatment for two and one-half years with a total of 80 gram seconds of beta radiation. *C* (M. W.), palpebral vernal conjunctivitis of two years' duration, and, *D*, same eye after nine treatments at intervals of two weeks with a total dose of 88 gram seconds of beta radiation.

Fig. 6.—*A* (I. M.), right eye, showing tuberculous sclerokeratitis; *B*, same eye two weeks after contact therapy to three areas, with 5 gram seconds of beta radiation to each area; *C*, same eye six weeks after contact and spray therapy. *D* (J. V.), deep central tuberculous keratitis; *E*, same eye after spray therapy with a total of 40 gram seconds of beta radiation.

Fig. 9.—*A* (J. D.), angioma vasculosum; *B*, appearance of the eye after contact therapy with 8 gram seconds of beta radiation. *C* (H. C.), bulbar papilloma (benign); *D*, complete disappearance of the lesion shown in *C* after spray and contact therapy with 70 gram seconds of beta radiation.

palpebral lesions, 12 limbal lesions and 7 a combination of palpebral and limbal lesions. Patients were considered cured if no signs or symptoms remained after treatment and if there was no recurrence the next year. Patients were said to show improvement when both subjective and objective improvement occurred but a few granulations or slight discharge persisted. Patients whose conjunctivitis was apparently healed after treatment and who did not return for a check the second season were placed in the group showing improvement. Patients were considered to show no improvement when severe symptoms continued and the follicles persisted after adequate therapy.

With the lesions of beta ray therapy, 30 per cent of the 60 patients healed; 60 per cent showed improvement, and 7 per cent showed no improvement.

In early cases of vernal conjunctivitis the papillae consist chiefly of proliferating young fibrous tissue, new vessels and cells of the lymphoid series. These elements are all sensitive to irradiation. In cases of

TABLE 2.—*Results of Beta Radiation Therapy for Vernal Conjunctivitis*

	Total Number	Cure		Condtion Improved		Condition Unimproved	
		Number	Per Cent	Number	Per Cent	Number	Per Cent
Palpebral	41	8	20	31	75	2	5
Limbal	12	5	42	6	50	1	8
Combined	7	5	71	1	14.5	1	14.5
	60	18	30	38	63	4	7

long-standing disease the papillae contain hyaline degeneration, which, as Pendergrass pointed out, is relatively insensitive to irradiation and explains the less dramatic results in these cases.

Patients with limbal and combined lesions were usually brought to the clinic at an earlier stage in the disease than patients with the palpebral variety, and the lesions thus responded more readily to treatment. In only 1 case (C. O.) was the limbal lesion of long standing, and it showed the same resistance to therapy as the long-standing palpebral lesions. The slightly better results for the combined group are not significant, owing to the small number of cases.

TURERCULOSIS OF THE ANTERIOR OCULAR SEGMENT

Encouraging results in the treatment of tuberculosis of the anterior ocular segment with beta radiation were reported by Woods [9] in 1939.

Material.—Data on 53 patients, representing 72 eyes treated since that time, are given in table 3. The duration of the disease before treatment had ranged from one week to as long as thirty years. All the cases were characterized by remissions and

9. Woods, A. C.: Treatment of Tuberculosis of the Anterior Portion of the Eye with Beta Rays of Radium, Arch. Ophth. **22**:735 (Nov.) 1939.

TABLE 3.—*Data on Patients with Tuberculosis of the Anterior Ocular Segment*

Name	Eye Affected	Duration Before Treatment	Vision Before Treatment	No. of Treatments and Interval *	Total Dose, Gm. Sec.	Final Vision	Final Clinical Picture and Period of Observation
D. L.	R. E.	18 yr.	20/30	34 in 6 yr.	223.5	20/50	Healed 2 yr.
	L. E.	18 yr.	20/15	13 in 6 yr.	88	20/15	Healed 6 yr.
E. G.	R. E.	1 yr.	20/20	5 courses, 3 wk. intervals	93.4	20/15	Healed 1 yr.
	L. E.	1 yr.	20/20	3 courses, 3 wk. intervals	32	20/15	Healed 1 yr.
M. D.	R. E.	12 yr.	Hand movements	3, weekly	16.2	Hand movements	Healed 2½ yr.
E. Y.	R. E.	1 mo.	20/20	3, weekly	15.3	20/15	Healed 1 yr. 4 mo.
E. H.	L. E.	1 yr.	20/30 —1	3, weekly	25.5	20/20	Healed 1 yr.
A. R.	R. E.	3 yr.	20/30	5, monthly	33.3	20/30 +4	Healed 2 yr.
	L. E.	3 yr.	20/70	5, monthly	33.3	20/30 —3	Patient wrote of flare-up 2 yr. after treatment; eye improved
M. M.	R. E.	5 yr.	20/200	15 in courses	86	20/100	Improvement; 2 slight flare-ups in next 2 yr.
	L. E.	5 yr.	6/200	14 in courses	82.2	20/60	Improvement; patient not seen after discharge in 1940
G. S.	R. E.	30 yr.	Hand movements	1941: 6	43.6	20/70	Improvement; 1 flare-up in 2 yr. after treatment
	L. E.	30 yr.	20/30	1942: 6	31.1	20/20	Healing; patient followed 2 yr.
E. M.	Both	20 yr.	20/30 +3	1937: 1	16.9	20/40	Healing; follow-up for 2 yr.
			20/15	1940: 3	24	20/15	Improvement; patient wrote of flare-up 5 yr. later
E. M.	R. E.	5 yr.	20/30	3, weekly	31.3	20/30	Improvement; relapse 1 mo. after discharge
H. R.	R. E.	2 yr.	20/15	16 in 2 yr.	157.3	20/15	Improvement; flare-up twice when treatment stopped; now on maintenance therapy
	L. E.	2 yr.	20/15	8 in 1½ yr.	57	20/15	Improvement; patient followed 2 yr.
C. H.	R. E.	5 yr.	20/30	1942: 15	151 total	10/200	No improvement; secondary glaucoma; cyclodiathermy
	L. E.	5 yr.	20/70	1938-1939: 18	228 over periods	20/40	Improvement for 5 yr., then flare-up; left eye quiet with therapy. 1 yr. eye quiet
E. M.	L. E.	9 yr.	5/200	1938: 9 courses	Exact dosage not known†	Eye enucleated	Improvement; then downhill course
R. M.	R. E.	8 wk.	10/200	3, over 2 areas in 72 wk.	30	20/30	Healed; follow-up 1 yr.
E. M.	R. E.	7 wk.	20/30	4, 2 courses	45	20/15	Healed; follow-up 1 yr.
E. C.	L. E.	1 wk.	20/50	2, 1 wk. interval	20	20/30	Healed; follow-up 6 mo.
I. M.	R. E.	3 mo.	20/30	3 treatments 5 Gm. sec. each to 3 areas	15, contact and spray	20/30 +4	Healed; follow-up 1 yr.
F. M.	L. E.	16 yr.	20/30 —3	3 courses over 3 yr.	1938: 38 1941: 20 1944: 8	20/50; senile immature cataract	3 recurrencies; response each time to irradiation; now healed 1 yr.

* The numeral not otherwise qualified indicates the number of treatments.
† Treated at the Kelly Hospital.

TABLE 3.—*Data on Patients with Tuberculosis of the Anterior Ocular Segment—Continued*

Name	Eye Affected	Duration Before Treatment	Vision Before Treatment	No. of Treatments and Interval *	Total Dose, Gm. Sec.	Final Vision	Final Clinical Picture and Period of Observation
X.	R. E.	1 mo.	20/30 —2	4	19	20/30	Healed; follow-up 1 yr.
B. M.	R. E.	6 yr.	20/100	7, 3 courses	66	20/100	Improvement, 1 yr.
	L. E.	6 yr.	20/200	to each eye	66	20/100	Improvement, 1 yr.
P. M.	R. E.	1 mo.	20/20	1	12	20/20	Improvement; eye quiet 6 mo. to date
M. V.	L. E.	Many times in 20 yr.	5/200	3 courses	65	20/70 (pin hole)	Improvement; eye quiet now 10 mo., to date
A. R.	L. E.	6 wk.	Hand movements 2 ft.	7, 2 wk. intervals	68	20/20	Healed 1 yr.
A. B.	R. E.	3 wk.	20/30	4	32	20/30	Improvement; follow-up 6 mo.
R. P.	R. E.	5 days	20/40	4	23.3	20/15	Healed; follow-up 3 yr.
H. G.	R. E.	1 yr.	20/30	5, 1 course 1941 and 1942	40.6	20/20	Healed; follow-up 2 yr. 9 mo.
	L. E.	5 yr.	20/15	5, 1 course 1941 and 1942	59.5	20/30	Healed; follow-up 2 yr. 9 mo.
M. P.	R. E.	2½ yr.	20/50	16 in 2 yr.	83.1	20/50	Healed; follow-up 4 yr.
	L. E.	2½ yr.	20/50	16 in 2 yr.	68.3	20/15	Healed; follow-up 4 yr.
J. O.	L. E.	5 yr.	Light perception; cataract	5 weekly then maintenance dose at 2 mo. intervals	75	Light perception	Healed; follow-up 3 yr.
A. R.	L. E.	3 mo.	20/200	13 in 14 mo., in courses	100.1	20/40	Healed; follow-up 2 yr.
N. C.	R. E.	10 yr.	Hand movements cataract	13, in courses	80	Hand movements	Healed; follow-up 3 yr.
M. L.	R. E.	1 yr.	20/15	4	40	20/15	Healed; follow-up 2 yr.
	L. E.	1 yr.	20/70	4	40	20/15	Healed; follow-up 2 yr.
J. V.	R. E.	1 mo.	20/15 —3, immature cataract	6	44	20/40	Healed; follow-up 1 yr.
	L. E.	1 mo.	Count fingers, 2 ft.	6	40	20/40	Healed; follow-up 1 yr.
H. C.	R. E.	4 yr.	20/70 +1	3	18.3	20/30	Healed; follow-up 3 yr.
	L. E.	6 yr.	20/20	3	18.3	20/15	Healed; follow-up 3 yr.
E. C.	L. E.	1 mo.	1/200	4	28.4	20/30	Improvement; follow-up 10 mo.
J. S.	R. E.	8 yr.	5/200	3 (both eyes)	25.5	20/70	Improvement; follow-up 9 mo.
	L. E.	8 yr.	Count fingers 6 ft.		25.5	20/70	Improvement; follow-up 9 mo.
P. D.	R. E.	1 mo.	Hand movements	14 over 8 mo.	137.2	20/30 +2	Improvement; follow-up 9 mo.
J. B.	R. E.	5 yr.	20/70	1938: 11 1942: 13	212	20/40	Improvement; 1 relapse; patient followed 7 mo. after
M. L.	R. E.	1 mo.	20/30 —2	3	15	20/30	Healed, 1 yr.
M. D.	.....	1 mo.	2/200	1937: 4	Exact†	20/20	Healed, 3 yr.
		14 yr.	20/20	1940: 2	16:7	20/20	Improvement; patient not seen after discharge

TABLE 3.—*Data on Patients with Tuberculosis of the Anterior Ocular Segment—Continued*

Name	Eye Affected	Duration Before Treatment	Vision Before Treatment	No. of Treatments and Interval *	Total Dose, Gm. Sec.	Final Vision	Final Clinical Picture and Period of Observation
Y.	L. E.	5 yr.	Hand movements 3 ft.	8 with 2 wk. intervals	51.5	20/15	Healed, 3 yr.
A. J.	R. E.	2 mo.	4/200	8 with 2 wk. intervals	88	20/40	Healed, 1 yr.
E. M.	R. E.	1 yr.	10/200	1938: 7 1939: 1	57.29	20/100	Improvement; 1 relapse, which responded to treatment
C. D.	R. E.	10 yr.	20/50, immature cataract	41 over 3 yr.	173.7	20/20	Improvement; number of small exacerbations, controlled with irradiation
	L. E.	10 yr.	20/200 immature cataract	17 over 3 yr.	79.4	20/100	Improvement
M. S.	R. E.	5 yr.	20/70	25 over 3 yr.	136	20/50	Improvement; 2 flare-ups in 3 yr.
	L. E.	5 yr.	5/200	25 over 3 yr.	136	20/200	Improvement; 2 flare-ups in 3 yr.
A. T.	R. E.	1 mo.	3/200	3, weekly; 10 maintenance doses	84	20/50 —1 (pin hole)	Healed, 2 yr.
I. T.	R. E.	10 yr.	20/30	1941-1942: 3	37.3	20/70	No improvement
	L. E.	10 yr.	Count fingers	1941-1942: 3	37.3	Counting fingers	No improvement
A. S.	R. E.	10 yr.	20/40	30, weekly, then monthly	146.9	20.70	No improvement; follow-up 2 yr.
	L. E.	10 yr.	20/40	29, 1 course, then monthly	131.1	Light perception	No improvement
A. D.	R. E.	4 mo.	20/200	3	17.2	20/200	Healed; follow-up 4 yr.
E. R.	L. E.	6 yr.	20/50	7	39.9	20/30	Healed, 1 yr.
M. C.	R. E.	2 mo.	20/40	4	36	20/40	Healed; follow-up 1½ yr.
J. E.	R. E.	1 mo.	20/40	4	30	20/30	Improvement, 6 mo.
H. C.	R. E.	3 mo.	20/20	3	25	20/20	Improvement, 6 mo.

exacerbations. A few of these patients had been followed in the Wilmer dispensary for ten to fifteen years, and from the data on these patients an attempt was made to evaluate the length and severity of exacerbations with and without beta therapy. In any such problem controls are extremely difficult. In the over-all picture, however, it seemed clear that treatment shortened the course of the attack and lessened its severity.

In 1 instance it was possible to treat one eye and use the other as a control. The patient, A. R., had severe bilateral central keratitis, which was at first thought to be due to congenital syphilis. However, intensive penicillin therapy produced no improvement, and the lesion gradually assumed the appearance of tuberculous keratitis (fig. 4 *A* and *B*). Beta irradiation was started on the more severely affected left eye on Dec. 28, 1944. Seven applications to the limbus at intervals of two weeks, using the spray technic, with a total of 52 gram seconds, were given. Both eyes remained intensely inflamed for eight weeks. By the tenth week the right eye was almost quiet; the left eye still showed slight pericorneal congestion, but its cornea was clearer than that of the right eye. At the end of four months the treated (left) eye was the better one, with less deep scarring and vascularization (fig. 4 *C* and *D*). Figure 5, a drawing of the right eye made with the slit lamp, shows the corneal nebulas and deep vessels, which were not present in the treated (left) eye.

An example of improvement in milder lesions is the case of I. M., a woman aged 50, who was first seen Oct. 20, 1944 with typical sclerokeratitis of the right eye of three months' duration. She had been hospitalized for one month by her local physicians for treatment with typhoid vaccine and sulfonamide compounds, and the usual local treatments had been employed, without improvement. Figure 6 *A* shows this eye before treatment; figure 6 *B*, the eye two weeks after the first treatment, in which a dose of 5 gram seconds was given by contact in three areas, at the site of the scleral inflammation and at the limbus from 3 to 6 o'clock. Symptoms were notably reduced after the first treatment and disappeared completely after the second treatment, in which 5 gram seconds of radiation was sprayed over the limbus at 3 to 5 o'clock. A third spray treatment with 5 gram seconds of radiation was given in the sixth week. When checked, six weeks after the last treatment, the eye had

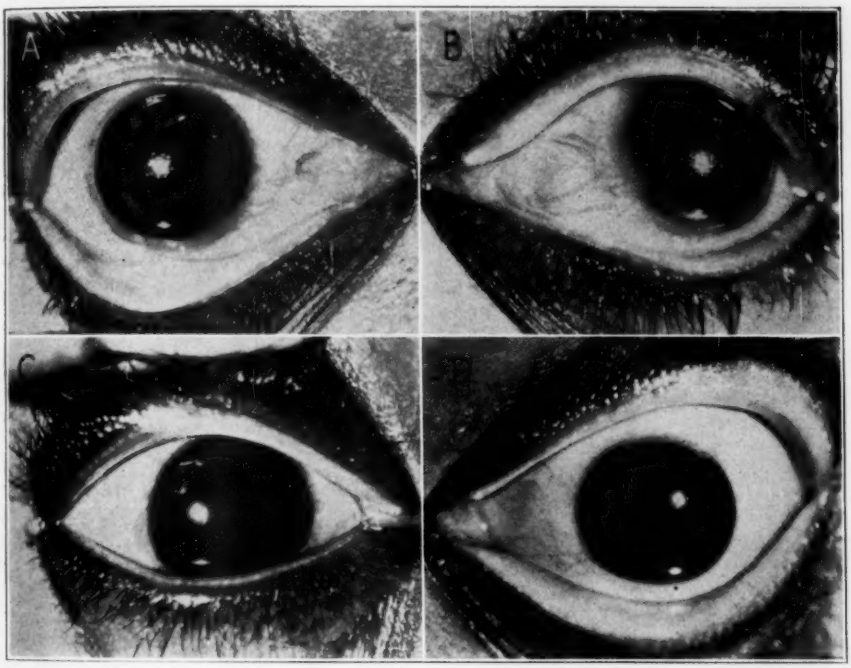

Fig. 4 (A. R.).—*A*, right eye (untreated control) and, *B*, left eye, with deep central tuberculous keratitis; *C*, right eye at the end of twelve weeks, without treatment; *D*, left eye, after treatment for twelve weeks with 52 gram seconds of beta radiation administered by spray to the limbus.

been asymptomatic for nine weeks, and the lesion had remained clinically unchanged for five weeks (fig. 6 *C*). Vision was 20/30 before and after treatment. The eye has remained quiet for one year, to date.

Another patient, J. V., had had violet keratitis (fig. 6 *D*) for three months. The sclerosing of the superficial vascular trunks by irradiation, with clearing of the cornea as the active process quieted down, is seen in figure 6 *E*. The total dose administered by the spray technic was 40 gram seconds, given in two courses. Vision improved from counting fingers at 2 feet (30 cm.) to 20/40.

Results.—Table 4 gives the results of treatment for the 72 eyes with tuberculosis of the anterior ocular segment. Lesions were considered

healed if the eye was quiet, with no corneal infiltrates, aqueous flare, nodules on the iris or keratitis punctata. These eyes were all kept under observation for at least a year. In 7 eyes the aforementioned criteria for healing were fulfilled except that the period of observation was less than one year. These 7 eyes are, therefore, placed in the group showing improvement. Eyes were considered to show improvement if after therapy they were asymptomatic and showed no more than residual conjunctival hyperemia, no aqueous ray and inactive corneal infiltrates. Eyes classified as unimproved showed no alleviation of symptoms and a progression of the disease in spite of adequate therapy. Of the 72 eyes, the lesions of 52.8 per cent were considered healed after treatment; the condition of 38.9 per cent was improved, and that of

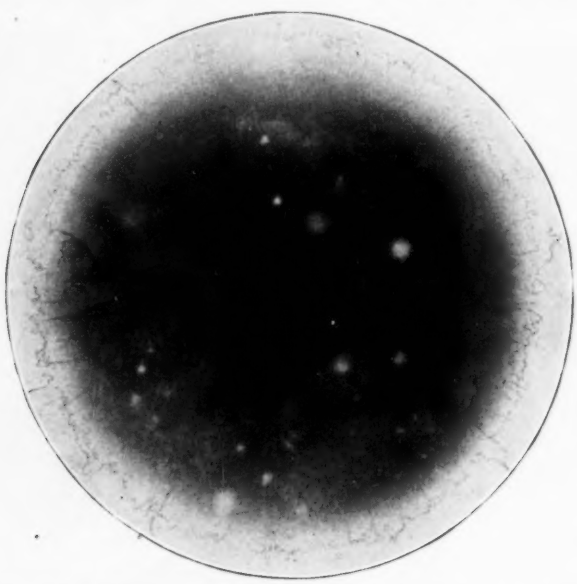

Fig. 5 (A. R.).—Slit lamp drawing of the right eye, as a control, showing residual nebulas and vascularization, which are not present in the left (treated) eye.

8.3 per cent showed no improvement in spite of adequate therapy. Since any clinical classification, such as "healed," "improved" and "unimproved" presents obvious difficulties, a comparison of vision before and after treatment is especially interesting (table 5).

Vision was improved two lines or more in 42 per cent of the eyes; maintained in 46 per cent and decreased in 9 eyes, or 12 per cent. The decrease in vision in 2 of the 9 eyes was due to the progress of senile cataracts.

Comment.—A comparison of the clinical and visual results is interesting, as it shows a close correlation between the total number of eyes in which the lesions were considered healed and improved and the total

number of eyes in which vision was either improved or maintained. This suggests that the clinical observation of amelioration of symptoms and shortening of the duration of the disease were indications of an actual decrease in severity of the lesion, with less permanent damage to the eye.

In general, active treatment was continued as long as improvement occurred. Healing occurred in 1 case after a total of 31.9 gram seconds of beta radiation had been administered; in another case 223 gram seconds of radiation was given over a four year period, during which time there were two exacerbations. This eye has now been healed for two years.

As a rule, in the treated eyes subjective improvement preceded objective improvement by about two weeks. Objective improvement

TABLE 4.—*Results of Beta Irradiation of Seventy-Two Eyes with Tuberculosis of the Anterior Segment*

	Healed		Improvement		No Improvement	
Eyes	Number	Per Cent	Number	Per Cent	Number	Per Cent
72	38	52.8	28	38.9	6	8.3

TABLE 5.—*Comparison of Initial and Final Vision in Seventy-Two Eyes with Tuberculosis of Anterior Segment Treated with Beta Radiation*

	Improved		Maintained		Decreased	
Eyes	Number	Per Cent	Number	Per Cent	Number	Per Cent
72	30	42	33	46	9*	12

* The decrease in vision in 2 of the 9 eyes was due to the progress of senile cataracts.

was usually noted in about four weeks, and eyes were either quiet or greatly improved in six to eight weeks. With extremely severe lesions longer treatment was required.

While beta irradiation seemed to have a beneficial effect on the immediate attack, it did not prevent future attacks. On this basis, long term therapy in the form of tuberculin treatment was advised in all cases.

The mode of action of radiation in cases of ocular tuberculosis is unknown. It has been pointed out that both beta [10] and grenz [11] rays increase the antibody reaction in the cornea, and it may be that in this way the beneficial results are obtained.

10. Moore, J. I.: Increase in Antibody Production in Cornea with Beta Rays, paper read at a meeting of residents of the Wilmer Ophthalmological Institute, Johns Hopkins Hospital, April 1946.

11. Thompson, R.; Pfeiffer, R., and Gallardo, E.: Stimulation of Local Antibody Formation in the Cornea by Grenz Rays, Proc. Soc. Exper. Biol. & Med. 36:179, 1937.

TABLE 6.—*Data on Patients with Benign Tumors Treated with Beta Radiation*

	Name, Age	Lesion	No. of Treatments and Dosage	Result
Angioma	J. G. 3 mo.	Nevus vasculosus of caruncle, upper and lower cul-de-sacs and nasal portion of left upper lid, causing partial ptosis	5 treatments over 6 mo.; 42 Gm. sec. total dose to different areas in contact with lesion	Slow resolution of lesion
	A. Z. 4 yr.	Angioma cavernosus; bluish discoloration and thickening of skin of right upper lid with partial ptosis	3 treatments 3 wk. apart, totaling 19.6 Gm. sec.; contact with lesion on both sides of lid	Complete resolution within 2 mo.
	H. L. 5 mo.	Angioma vasculosum of left upper lid and left hand	4 treatments over 5 mo., total dose 40 Gm. sec., given just short of contact with lesion	Complete resolution in 5 mo.
	G. P.	Small mulberry angioma, bulbar conjunctiva	2 treatments, 14 Gm. sec. each	Lesion completely disappeared in 2 mo.
	Mrs. G.	Angioma of caruncle	2 treatments, 6 Gm. sec. each	Lesion completely disappeared in 4 wk.
	J. D.	Small angioma cavernosum, inner canthus	1 treatment, 8 Gm. sec. each	Disappeared in 9 wk.
Papilloma of lids	J. K.	Small, pedunculated papilloma on margin of right upper lid	1 treatment, 14.3 Gm. sec.	Tumor fell off in 2 wk.
	J. W.	Papilloma, margin of right lid	1 treatment, 6.9 Gm. sec.	Tumor fell off in 3 wk.
	H. W.	Papilloma, upper margin of lid	2 treatments, 9.3 and 7 Gm. sec., 1 wk. apart	Lesion disappeared in 1 mo.
	J. N.	Small papilloma, left lower lid	10 and 9.75 Gm. sec. 1 wk. apart	Lesion disappeared in 2 wk.
	Mrs. B.	Flat papilloma, right upper lid	8 Gm. sec., by contact	Lesion size in 3 wk.; patient became ill and could not return for further therapy
	S. C.	Flat papilloma, upper lid	4 treatments, 3 wk. intervals; 7, 9, 6 and 8 Gm. sec., respectively	Lesion did not change; treatment discontinued
	Mrs. W.	Pedunculated	1 treatment, 10 Gm. sec.	Lesion disappeared in 2 wk.
	Mrs. R.	Flat	3 treatments, 10 Gm. sec.	Lesion flattened in 2 mo.
	Miss P.	Flat papilloma, right upper lid	3 treatments, 6 Gm. sec. each	Lesion did not respond, surgically removed; diagnosis of nevus on section
Papilloma of caruncle	P. U.	Papilloma of caruncle	1 treatment, 7 Gm. sec.; contact	Lesion disappeared in 2 wk.
	F. R.	Papilloma of caruncle	3 treatments, 26 Gm. sec. in contact with lesion	Lesion disappeared in 1 mo.
	M. W.	Papilloma with granulation tissue at caruncle	7.9 Gm. sec., in contact with lesion	Lesion flattened in 4 wk.
Papilloma of bulbar conjunctiva	D. B.	Papilloma of upper cul-de-sac	3 treatments 2 wk. apart, in contact with lesion; doses 10, 15 and 12 Gm. sec.	Lesion disappeared in 2 mo.
	H. C.	Papilloma, bulbar conjunctiva (left eye)	8 treatments, total 75 Gm. sec.	Lesion completely disappeared; vision 20/30 when seen 2 yr. later
	M. S.	Papilloma, bulbar conjunctiva (left eye)	4 treatments, each in contact with lesion in 4 areas; total dose 57 Gm. sec.	Lesion completely disappeared in 2 mo.; vision 20/20 3 yr. later; no recurrence

TABLE 6—*Data on Patients with Benign Tumors Treated with Beta Radiation —Continued*

Name, Age	Lesion	No. of Treatments and Dosage	Result
W. G.	Papilloma partially covering both corneas; lesion in right eye surgically removed, base irradiated; left eye irradiated only	R. E.: 2 treatments, total 7 Gm. sec. L. E.: 5 treatments, total 40 Gm. sec.	Both lesions completely disappeared
A. G.	Papilloma marking left cornea	5 contact treatments, total 45 Gm. sec.	Lesion cleared and remained clear, as patient wrote 1 yr. later
D. S.	Papilloma at limbus early	3 treatments; total 32 Gm. sec.	Disappeared in 8 wk.; patient followed 8 mo.

BENIGN TUMORS OF THE LIDS AND CONJUNCTIVA

Removal of small benign tumors of the lids and conjunctiva is usually carried out for cosmetic reasons; thus, a form of therapy which will produce the least residual scar is important. In some cases the surgical approach is more expedient and more satisfactory; in others irradiation is more desirable. It is important for the ophthalmologist to differentiate between benign and malignant lesions, and in case of any doubt a biopsy should be done. Most malignant lesions require deeper and more intensive therapy and should be referred to the radiologist.

The data on 24 cases of benign tumors of the lids and conjunctiva are given, with the dose and the length of time necessary to effect a disappearance of the lesion (table 6). Angiomas as a rule respond well to irradiation, except the nevus flammeus, or port-wine stain. In contrast, the nevus vasculosus, or strawberry mark, frequently seen at the caruncle or involving the cul-de-sacs, responds dramatically.

J. G., a 3 month old baby, had a lesion involving the caruncle, nasal upper and lower cul-de-sacs and the nasal portion of the left upper lid (fig. 7 *A*). Five treatments, with a total dose of 42 gram seconds, administered in contact with the lesion were given over a period of six months. With this therapy there was progressive improvement (fig. 7 *B*).

A 4 year old boy (A. Z.) had thickening of the lid and partial ptosis, due to angioma cavernosum. This lesion is usually completely subcutaneous and consists of dilated vessels which produce an elevation and give a bluish color to the skin (fig. 8 *A*). Five months later, after three treatments, with a total of 19.8 gram seconds, his condition was much improved (fig. 8 *B*). The response with this type of lesion is excellent but not so dramatic as that with nevus vasculosis.

Figure 9 *A* shows a small angioma vasculosum near the inner canthus in a 4 year old girl, J. D. One contact treatment with 8 gram seconds of beta radiation caused complete resolution of the lesion (fig. 9 *B*).

Papilloma of the bulbar conjunctiva presents the most frightening picture of the benign tumors. Its growth is usually rapid, and it fre-

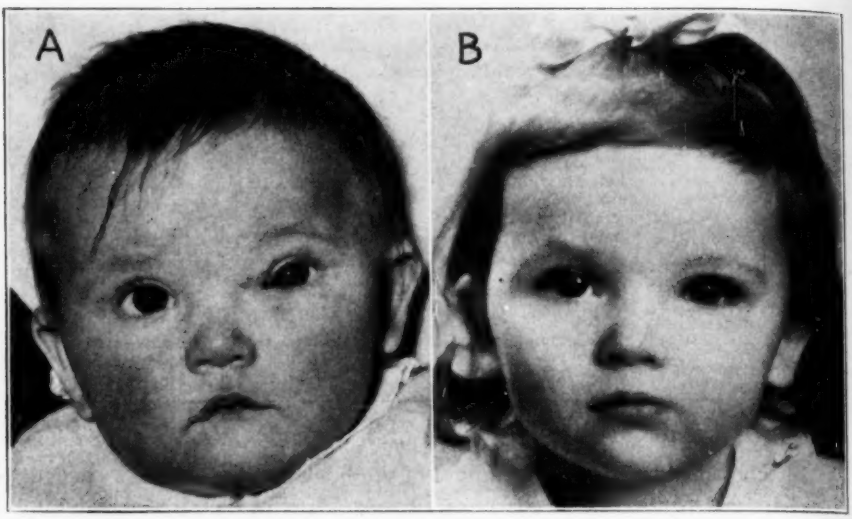

Fig. 7 (J. G.).—*A*, angioma of the caruncle and the upper and lower cul-de-sacs; *B*, appearance of the eye one year after therapy with 42 gram seconds of beta radiation.

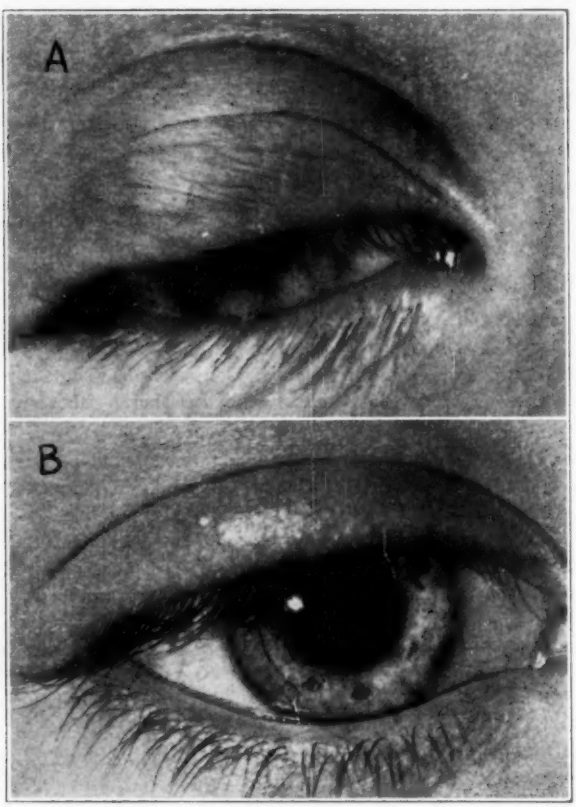

Fig. 8 (A. Z.).—*A*, angioma of the upper lid in a 5 year old boy; *B*, appearance of the eye five months after contact treatment with 19.8 gram seconds of beta radiation.

quently extends so far over the cornea that it affects the vision. In cases of this tumor biopsy is routinely done to rule out epithelioma. Both the benign and the malignant forms, however, respond well to beta radiation therapy.

Figure 9 *C* shows a typical bulbar papilloma of eight months' duration in a man aged 60. Thirty gram seconds of radiation given by spray and contact made the lesion almost completely disappear, and the patient did not return for two years.

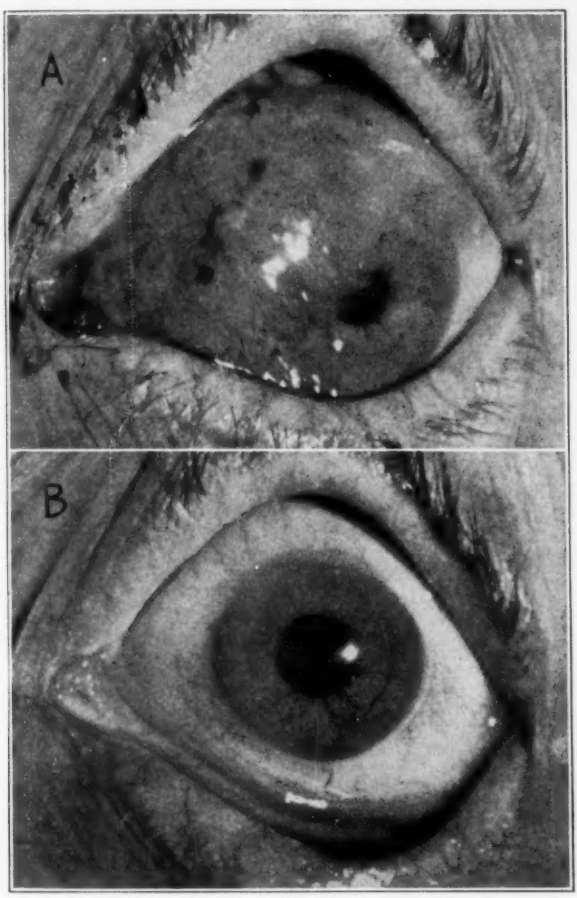

Fig. 10 (M. S.).—*A*, bulbar papilloma; *B*, appearance of the eye shown in *A* after ten weeks of contact and spray therapy with a total of 57 gram seconds of beta radiation.

At this time he had a mild recurrence and was given an additional 45 gram seconds as contact and spray therapy. The eye has remained quiet for three years, to date, with vision of 20/30 (fig. 9 *D*).

A woman aged 64, with a typical bulbar papilloma of three months' duration, was seen first on May 8, 1943 (fig. 10 *A*). Biopsy confirmed the clinical diagnosis. Contact treatment, consisting of a total of 57 gram seconds of radiation, was given

in four doses over a period of six weeks. In ten weeks the lesion had completely disappeared (fig. 10 *B*). The patient has been followed now for three and one-half years, and the eye has remained quiet, with vision of 20/20.

The pedunculated papillomas of the lids usually dry up after one, or at most two, treatments. One patient with this type of lesion is shown before treatment (fig. 11 *A*) and after contact therapy with a dose of 5 gram seconds (fig. 11 *B*).

Papillomas of the conjunctiva respond rapidly and leave little residual scar. D. B., a 10 year old Negro girl, had a mass in the upper cul-de-sac which on section proved to be a papilloma (fig. 12 *A*). Three contact treatments were given, for a total dose of 37 gram seconds, at two week intervals. The lesion two months later is shown in figure 12 *B*.

Fig. 11.—*A*, small papilloma of the lid; *B*, appearance of the eye after contact therapy with 5 gram seconds of beta radiation.

I wish to emphasize that the removal of benign tumors of the anterior ocular segment for cosmetic reasons is in the province of the ophthalmologist and that beta irradiation is practical and gives excellent results.

CORNEAL LESIONS

Punctate Keratitis.—Five patients with typical punctate keratitis which had responded to no other therapy were sent to the radium clinic for treatment. Notwithstanding the fact that punctate keratitis can be

produced by beta irradiation, 2 of these patients were given low doses, 3 to 5 gram seconds; 3 were given a full course of treatment, with 18 gram seconds. One patient in the latter group showed improvement and then relapsed. The other 4 patients showed no improvement whatever.

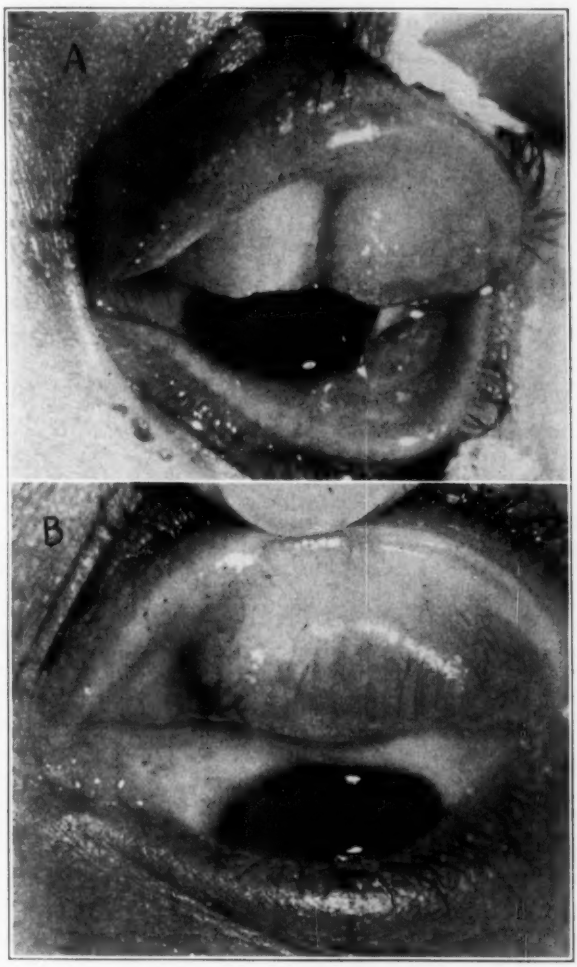

Fig. 12 (D. B.).—*A*, papilloma of the cul-de-sac; *B*, appearance of the eye after contact therapy with 37 gram seconds of beta radiation.

Corneal Ulcer.—Eleven patients with corneal ulcer, due either to staphylococcic or to mixed infection which had not responded to local therapy, were given one course of treatment. One patient showed notable improvement, but a marginal infiltrate shortly developed in the other eye. The picture became typically that of tuberculous ker-

atitis, which rapidly responded to radiation therapy. The infection in the first eye was probably also on this basis, with a superimposed secondary infection.

In 6 patients the treatment seemed to make the ulcer definitely worse, and 3 were admitted to the hospital. In 2 of these 3 patients conjunctival flaps were pulled, as the cornea became so thin that perforation was feared. The third patient seemed to improve with local therapy in the hospital, and the ulcer slowly healed. In 4 eyes with less

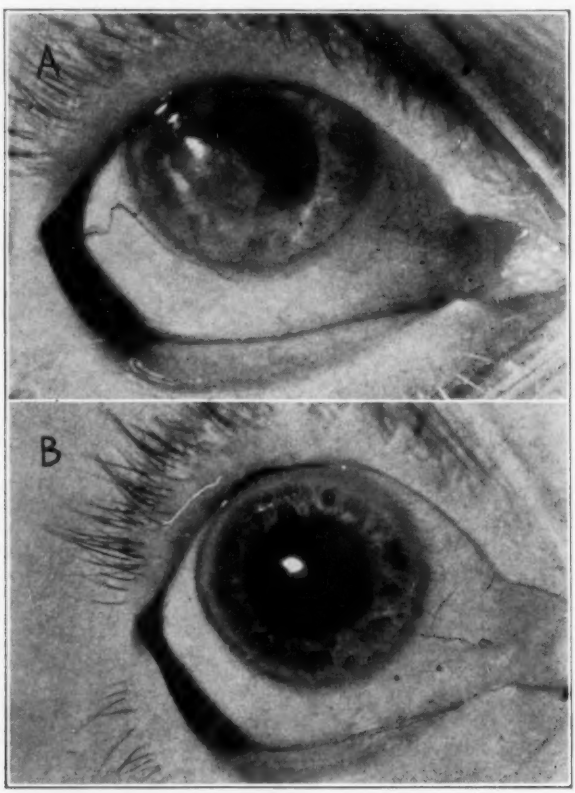

Fig. 13 (L. G.).—*A*, acne rosacea keratitis; *B*, appearance of the eye after three courses of spray and contact therapy, with a total of 45 gram seconds of beta radiation.

purulent discharge only questionable improvement occurred after the first course of treatment. However, after the discouraging experience with 3 patients who required admission to the hospital, a second course of therapy was not given. Here, again, the result is as would be expected, in that irradiation hastened the breakdown of the already damaged cells.

Chronic Ulcer.—Nine patients with corneal ulcer considered to have resulted from acne rosacea keratitis were treated. These ulcers consisted of stromal infiltrates, usually in the lower corneal quadrants, into

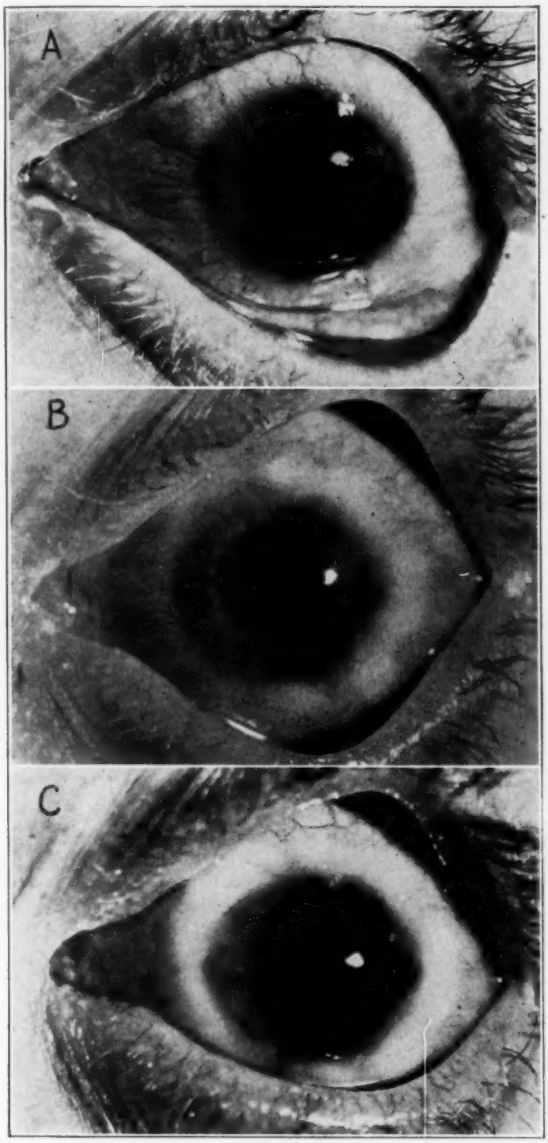

Fig. 14 (C. S.).—*A*, sulfur dioxide burns; *B*, temporal half of the limbus after treatment; *C*, the limbus after contact treatment with 5 gram seconds of radiation to each area.

which went deep and superficial vessels. The epithelium usually was intact, but on some occasions staining occurred. Treatment at the

limbus produced occlusion of the vessels, and with the decrease in vascularity the corneal lesions of 6 patients improved. In the 3 patients who showed no improvement the purulent discharge and conjunctival factor were prominent features.

The corneal lesion of L. G., a woman aged 50 with acne rosacea keratitis of six months' duration (fig. 13 *A*), received three courses of spray and contact therapy with a total dose of 45 gram seconds. In a

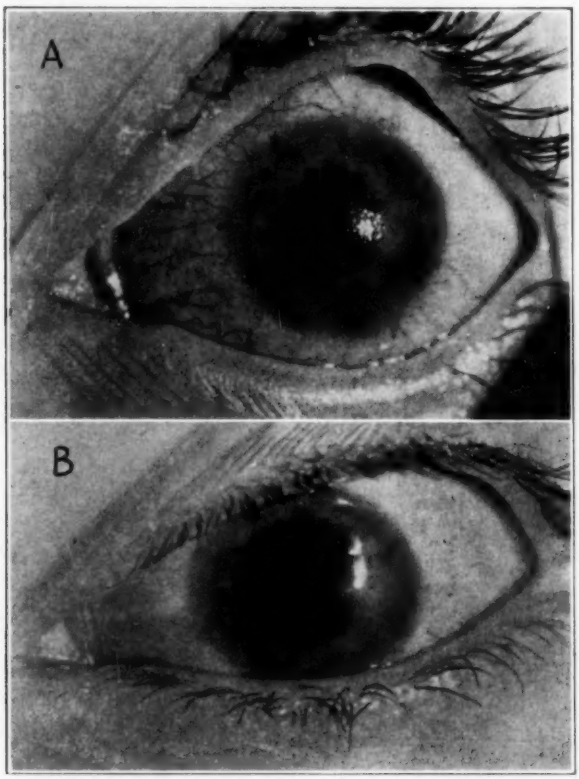

Fig. 15 (L. K.).—*A*, appearance of the eye after lamellar laminectomy, showing invading limbal vascularization; *B*, limbus treated with spray therapy only.

follow-up observation of two years there has been no recurrence (fig. 13 *B*).

Corneal Scars.—A great variety of corneal scars have been treated, and the results are extremely difficult to evaluate. Old dense scars or scars with deposits of calcium show no change, even after prolonged therapy.

Recent corneal lesions with thickening of the stroma and vascularization, on the other hand, do seem to show clearing with treatment, although here a control is difficult.

An illustrative case is that of C. S., whose left eye sustained a sulfur dioxide burn (fig. 14 *A*). During the past year many kinds of local treatment had been tried, without signs of improvement. Vision was reduced to 4/200 and was not improved with the pinhole disk. The eye was extremely painful, and there were intense photophobia and lacrimation. The temporal half of the limbus received 5 gram seconds of beta radiation by contact, and the vessels in this area were occluded (fig. 14 *B*). Vision improved to 20/200. Figure 14 *C* shows the nasal half of the limbus after treatment. Therapy was given at four week intervals, with a total

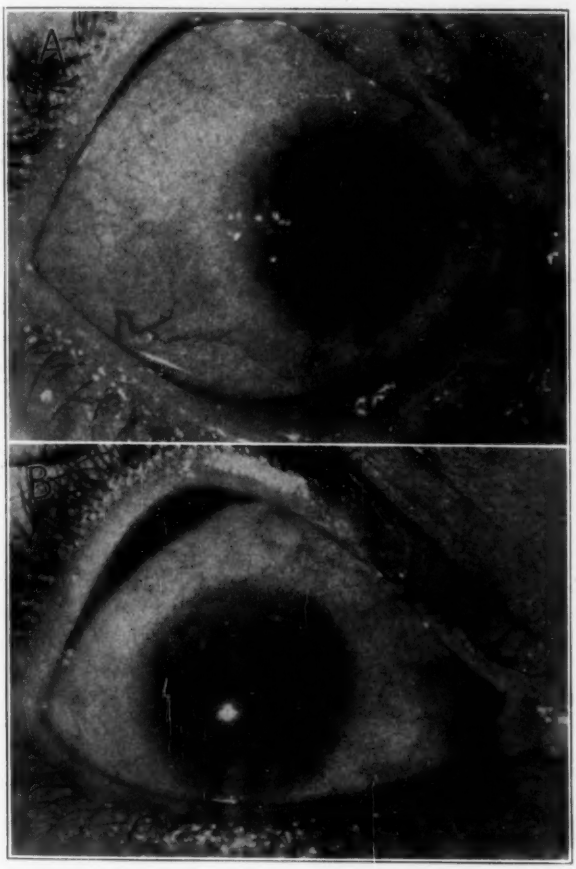

Fig. 16 (W. T.).—*A*, peculiar corneal lesion of unknown origin, in a Negro; appearance of the eye after treatment of 40 gram seconds of radiation to the limbus.

dose of 5 gram seconds administered by contact to each area and 10 gram seconds by spray. Vision one year later was 20/70; with the pinhole disk it was 20/50. The eye was asymptomatic.

In this case vascularization played a large part, and with beta irradiation it was possible to occlude the vessels. Young fibroblasts are sensitive to irradiation, and their activity is inhibited. In consider-

ing this feature, however, the relative sensitivity of the stromal cells is important; and to date we have no experimental evidence to show they are more resistant than the invading fibroblasts.

In cases of lamellar keratectomy routine treatment with beta rays is given to the limbus to prevent vascularization. The best results have been obtained with a course of treatment on the fifth to the seventh postoperative day, and again on the twenty-first day. The applicator is placed as close to the limbus as possible, and the circumference is treated by quadrants, using the spray technic. An attempt is made to confine

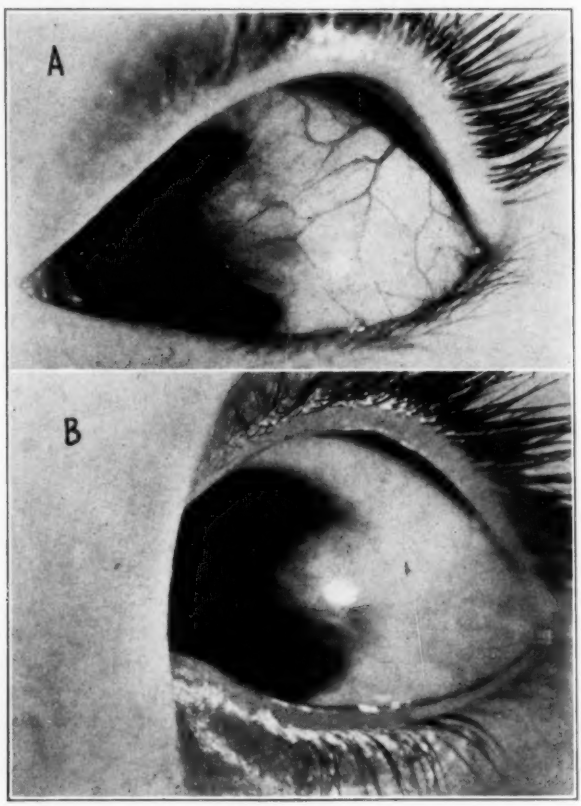

Fig. 17 (B. C.).—*A*, conjunctival flap; *B*, conjunctival flap after contact and spray therapy.

the treatment chiefly to the limbus and the conjunctiva, rather than to spread the rays over the new corneal epithelium. When irradiation is delayed until new vessels have already entered the cornea, larger doses are required to cause occlusion. This is produced most satisfactorily by contact treatment over the vessel at the limbus.

The eye of L. K., a woman aged 28, with fatty degeneration of the cornea is shown after lamellar keratectomy (fig. 15 *A*). After operation vascularization at the

limbus was controlled with beta rays (fig. 15 *B*). The vessels developed clubbed ends and did not extend farther.

After keratoplasty beta radiation is immediately used if there is any evidence of formation of new vessels or of old vessels opening up, for the superficial vascular process can in most cases be controlled by this method. Deep vessels are more difficult to treat, and in some instances it is necessary to use actual cautery. Corneal clouding due to other causes is not helped by irradiation, and treatment is contraindicated.

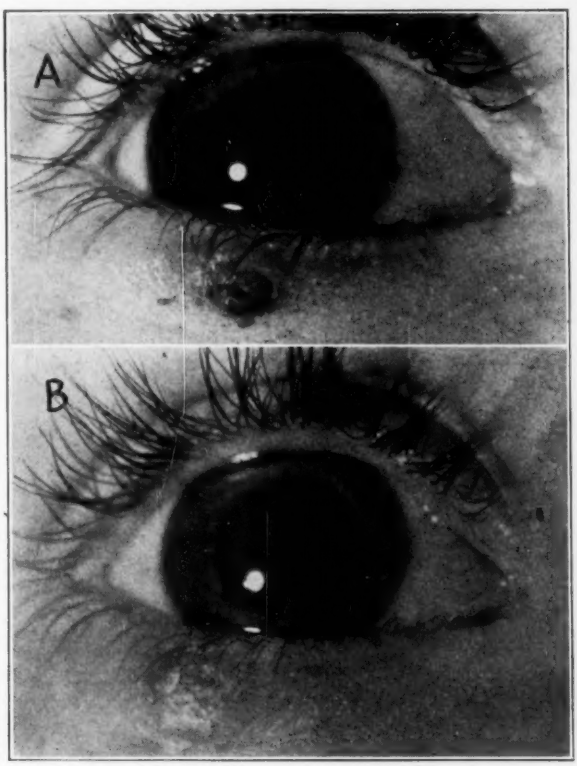

Fig. 18 (N. V.).—*A*, granulation tissue in a neglected chalazion; *B*, appearance after therapy with 10 gram seconds of beta radiation.

Figure 16 *A* shows a peculiar corneal vascularization which had persisted for nine months in a Negro aged 23. A complete survey of his case revealed no etiologic factor. He was treated with contact and spray irradiation to the limbus, with a total dose of 40 gram seconds. Figure 16 *B* shows the eye eight weeks later.

Pterygiums and Pseudopterygiums.—Eighteen patients with pterygiums were treated; in only 1 did treatment seem slow in yielding results, and operation was performed. In cases in which there is a recurrence after operation, beta irradiation is especially valuable. Contact therapy gives the more satisfactory results.

Conjunctival flaps over the cornea and granulation tissue can be flattened and made avascular.

B. C. had a severe corneal ulcer after an injury. A conjunctival flap was pulled over the cornea to save the eye. Irradiation was subsequently used to flatten the flap (fig. 17 *A* and *B*). In N. V., a child aged 7, a neglected chalazion produced a horn of granulated tissue on the cutaneous surface of the lower lid (fig. 18 *A*). One treatment, with 10 gram seconds, caused shrinkage and healing of the lesion (fig. 18 *B*).

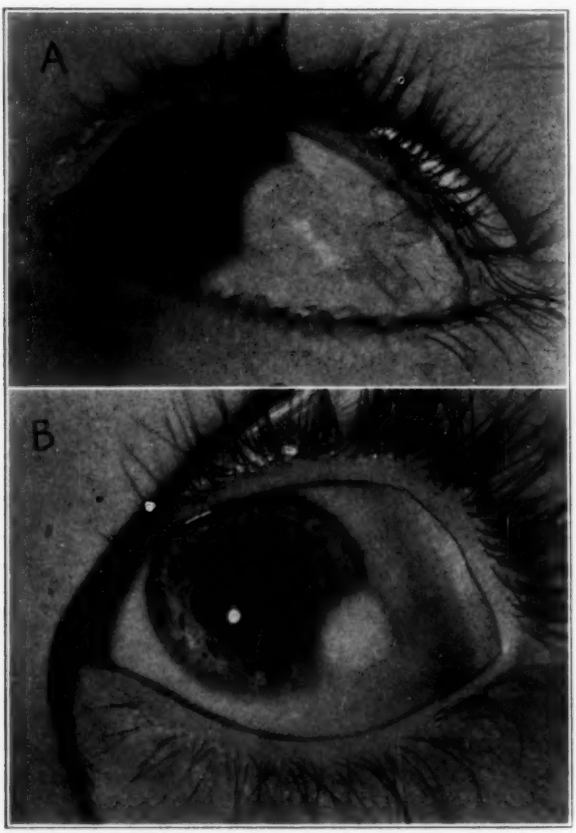

Fig. 19 (Kin).—*A*, dermoid of the limbus; *B*, dermoid after contact therapy with 15 gram second of beta radiation.

A dermoid at the limbus (fig. 19 *A*) after contact therapy with 15 gram seconds of beta radiation was flattened and made avascular (fig. 19 *B*).

Other Lesions.—A patient with blastomycosis of the outer canthus was successfully treated by contact and spray beta irradiation, with complete resolution of the lesion. Nine patients with sarcoid involving the cornea and iris were treated, without benefit. In patients with corneal dystrophies, lupus erythematosus and pemphigus, no improvement was noted after adequate therapy.

CONCLUSION

1. In the treatment of vernal conjunctivitis beta irradiation gives excellent results. Recurrences are less frequent after treatment, and when they do occur are less severe. Sixty patients were treated, with cure in 30 per cent, improvement in 63 per cent and no improvement in 7 per cent.

2. In treatment of tuberculosis of the anterior ocular segment beta irradiation seems to shorten the course and decrease the severity of the symptoms, with less residual scarring of the cornea. Irradiation, however, did not prevent future recurrences of tuberculosis.

3. Small benign tumors of the anterior ocular segment can be removed by beta irradiation easily and safely, with excellent cosmetic results. Since the method is extremely easy to use in an office and requires neither hospitalization nor general anesthesia, most patients prefer it to operation. These advantages make it particularly satisfactory for work with children.

4. In treatment of corneal scars in which vascularization is a prominent feature and there is still activity irradiation appeared to be beneficial. With old, calcified and dense scars treatment is not successful.

5. Beta irradiation is ideal for preventing superficial vascularization after keratectomy and keratoplasty.

6. Granulation tissue, conjunctival flaps and pterygiums can be made avascular with contact therapy.

7. Encouraging results in the treatment of acne rosacea keratitis, with limbal application to obliterate vessels, are obtained.

8. Corneal dystrophies, sarcoid, lupus erythematosus, pemphigus, punctate keratitis and corneal ulcers due to pyogenic infections are not benefited with beta rays, and may be made worse by their use.

Dr. James I. Moore treated some of the patients included in the group with ocular tuberculosis and permitted the use of these cases in the series.

Photographs were taken by Mr. Delbert Parker, of the Wilmer Ophthalmological Institute.

12 West Read Street (1).

A SCREENING TEST FOR DEFECTIVE RED-GREEN VISION

Test Based on Eighteen Pseudoisochromatic Plates from the American Optical Company's Compilation

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AND

M. CATHERINE RITTLER, B.A.

NEW YORK

PROBABLY the demand most frequently made of those who have had experience in testing for color blindness is for a simple means of detecting the presence of defective color vision—in other words, for a simple screening test adequate to differentiate between normal and defective color vision. The demand comes from the military services; from ophthalmologists; from industry, with its increasing use of color, and from vocational high schools, where too often a boy is trained for a trade only to find near the end of his training that he cannot meet the requirements for color vision. Tests have been produced which when properly administered and the critical scores established will meet this need, as was shown by us in previous reports [1]; but these tests are of foreign make and are not now available in quantity. Therefore, their evaluation has at present more theoretic than practical value. Other tests are under production which are planned both to detect the presence of deficiency in color vision and to analyze its type and amount. But lag in production affects the making of color tests as well as of automobiles, and it may be some years before these tests can be put into general use. In the meantime something must be found to serve as an adequate screening test of deficiency in color vision—something which is immediately available.

The only available test which gives promise of success for this purpose is the Pseudo-Isochromatic Plates for Testing Color Perception, compiled in 1940 by the American Optical Company for the military

From the Knapp Memorial Laboratories, Institute of Ophthalmology, Columbia University College of Physicians and Surgeons.

1. Hardy, L. H.; Rand, G., and Rittler, M. C.: (*a*) Tests for Detection and Analysis of Color Blindness: I. An Evaluation of the Ishihara Test, Arch. Ophth. **34**:295 (Oct.) 1945; (*b*) II. Comparison of Editions of the Ishihara Test, ibid. **35**:109 (Feb.) 1946; (*c*) III. The Rabkin Test, ibid. **35**:251 (March) 1946.

services. This compilation consists of 46 plates copied from either Ishihara's Test for Colour-Blindness or Stilling's Pseudo-Isochromatic Plates for Testing the Color Sense. Designed principally for the detection of deficiencies in red-green perception, it has been widely used, but with generally unsatisfactory results. In part this is because of inaccurate color reproduction and a poor selection of plates for the compilation; in part, because no critical scores are provided. That is, no information is given by the compilers of the test as to the number of plates likely to be failed by subjects whose color perception is normal or of the number likely to be passed by those whose color perception is defective.

In this report of our experience with the compilation administered as one of a battery of color tests given to a large group of subjects having normal, low normal or defective color vision of varying degrees of defect, we plan to show that this compilation of plates, even when properly administered under the quality of illumination accepted as standard for color testing,[2] is inadequate to detect with reasonable accuracy the presence of defective color vision. On the constructive side, we shall present a selection of 18 plates of this compilation which, when the test is properly administered, is adequate to screen persons with even minor degrees of defective red-green vision from those whose color vision is within normal limits.

As previously reported, our general procedure was to administer a large battery of color tests, both laboratory and clinical in type. On the basis of the composite picture obtained, we classified the color vision of each subject under the following categories: normal, low normal, low discrimination sufficient to amount to a color defect, anomalous trichromasy (protanomaly, deuteranomaly and tritanomaly) and dichromasy (protanopia, deuteranopia and tritanopia[3]). A description of the color vision typified by these terms has already been reported.[4] Our data thus permit us to evaluate each test of the battery as a diagnostic or screening medium, and as a medium for the differential classification of the type and extent of the color vision defect.

It is not our intention to present here a detailed analysis of the responses to each plate of the American Optical Company's compilation made by subjects representing each of the types of defective color vision, as we did for the Ishihara and Rabkin tests. There are no specific plates

2. Hardy, L. H.: Standard Illuminants in Relation to Color-Testing Procedures, Arch. Ophth. **34**:278 (Oct.) 1945.

3. We have had the opportunity to study too few cases of tritanomaly and tritanopia to warrant their inclusion in the present study. The experimental subjects reported on here have predominantly defective red-green vision.

4. Hardy, L. H.; Rand, G., and Rittler, M. C.: Color Vision and Recent Developments in Color Vision Testing, Arch. Ophth. **35**:603 (June) 1946.

in this compilation designed to analyze type of defect. We plan merely to show the distribution of errors made on the compilation for 365 subjects whom we judged on the basis of our battery of tests to have normal color vision and for 117 subjects similarly judged to have defective red-green vision. Our method of determining whether the color vision fell within normal limits has already been described.[5] The subjects tested were not selected on the basis of any statistical survey. They included patients referred to us by ophthalmologists, men rejected by the armed forces because of defective color vision and subjects obtained from some of the New York city high schools and from the Vanderbilt Clinic. Only the 40 plates designed to test literate subjects were scored. The demonstration plates (25, 26 and 46) and the "path" plates for illiterates (35, 36 and 45) were not included. For all testing the book was placed on the rack of the Macbeth easel lamp. This equipment, which was designed to furnish standard conditions for testing color blindness with pseudoisochromatic plates, provides 45 foot candles of illumination, the quality of which is the closest approximation to that of ICI Illuminant C commercially available.[2] The light fell on the test at an angle of 45 degrees; the viewing angle was normal, or 90 degrees, and the testing distance was approximately 30 inches (75 cm.).

THE FORTY PLATE SERIES

Results for the 40 plate series are given in figure 1. The data for the 365 subjects judged as having normal color vision are presented at the left of the chart, as rectangles in outline; the data for the 117 subjects with defective color vision, at the right of the chart, as black rectangles. The number of plates failed (error score), in increasing groups of 2, is plotted on the horizontal coordinate, and the percentage of subjects failing each group, on the vertical coordinate.

Normal Color Vision.—As can be seen in figure 1, only 12.5 per cent of subjects with normal color vision failed none of the plates or only 1; 46 per cent failed 6 or more plates; the maximum number of plates failed was 17. Rarely was there complete failure to see a digit, as occurs frequently when color vision is defective. Rather, the usual type of error was a misreading of digits due to lack of careful scrutiny, as *48* for *43* (plate 2); a misreading of Stilling digits of German formation, as *25* for *75* (plate 30); the reading of the digits supposed to be invisible to persons with normal color sense (plates 33 and 34), or the partial misreading of a digit of the Ishihara plates, as *20* for *29* (plate 11). It should be noted that the printing of those sections of the digit supposed to be seen only when red-green perception is defective appears in stronger

5. Hardy, Rand and Rittler,[1c] p. 257.

coloration than in the original plates. This is particularly true of the green-blue and blue portions of these digits. There is, therefore, a greater incentive for the normal subject to read them, or parts of them, as reproduced by the American Optical Company than as presented in the original Ishihara test.

It may seem strange to one who has not had a wide experience in administering this compilation to a large unselected group, varying in intelligence and interest, that so many errors of these types can be made by persons with normal color vision. This, however, has been the general experience of persons who have administered it in the military services, in industry and in schools. The compilation is definitely faulty

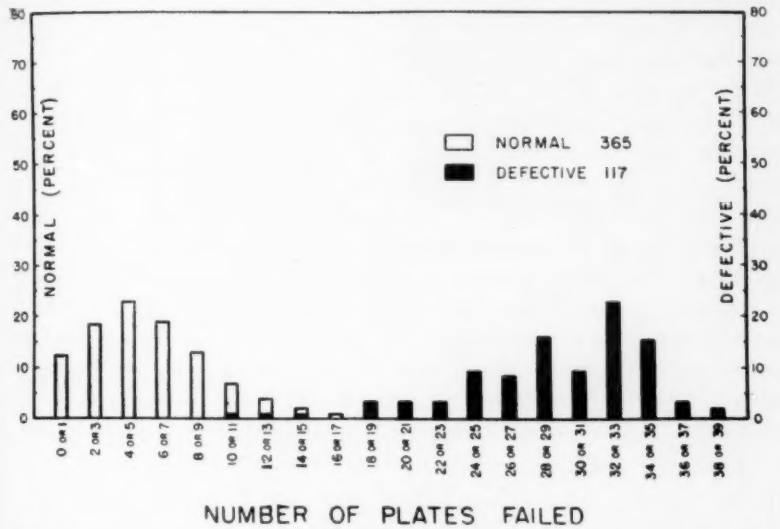

Fig. 1.—Results for the 40 plate compilation of the American Optical Company's pseudoisochromatic plates.

in its inclusion of so many plates subject to misreading because of factors other than defective color vision.

Defective Color Vision.—As can be seen in figure 1, subjects whose color vision was diagnosed as defective on the basis of our battery of tests failed as few as 10, and as many as 39, of the 40 plates used.

In brief, there is a range of 10 to 17 plates failed, which includes the error scores of some whose color vision was normal and some whose color vision was defective. Diagnosis on this compilation, therefore, is not possible when error scores occur within this range. In other words, no critical score can be established for this compilation which will differentiate between normal and defective color performance.

THE EIGHTEEN PLATE SERIES

The problem, however, cannot be abandoned at this point. This test must, if at all possible, be made to do the work, since, as stated earlier, it is the only one worth considering which is commercially available at the present time. We feel that the problem can be met fairly satisfactorily by a selection of 18 plates from the American Optical Company's compilation. In selecting each plate we were not guided solely by statistical criteria. Our aim was simply to select a series of plates that would yield a critical score for normal and defective red-green vision. In general, we discarded as far as possible those plates on which insignificant errors were made, owing to factors other than defective color perception. At the same time, we needed to retain some plates which were failed by subjects whose defect in color vision was mild in degree even if these plates were occasionally misread by members of the normal group. In other words, we could not sacrifice the sensitivity of the series to detect minor degrees of defective color perception in the interest of securing a perfect response from all whose color vision is within normal limits. Also, we wished to include some plates failed by persons whose defect in color vision was marked. These considerations barred us from searching for plates passed by all the normal group and failed by all the group with defective color vision. Further, attention had to be given to excluding plates which vary greatly from copy to copy of the compilation. For this reason, plates 39, 40, 43 and 44 were rejected from our selection. Fundamentally, the selection was a trial and error procedure, being adjusted and readjusted according to the responses given chiefly by those normal subjects who failed many plates and by those subjects with defective color vision who failed few plates.

Our final selection includes plates 3, 4, 5, 6, 8, 9, 12, 13, 16, 17, 19, 20, 21, 23, 27, 29, 41 and 42. The results are shown in figure 2 for 365 subjects whose color vision we judged to be within normal limits and for 150 subjects whose color vision we judged to be defective. As in figure 1, the data for the normal group are presented at the left of the chart as rectangles in outline; the data for the group with defective color vision, at the right, as black rectangles. The number of plates failed (error score) is plotted on the horizontal coordinate, and the percentage of cases failing each successive number of plates, on the vertical coordinate.

Normal Color Vision.—As can be seen in figure 2, 93 per cent of the subjects we judged on the basis of our battery of color tests to have normal color vision failed none of the 18 plates, or only 1; 4 per cent failed 2 plates; 2 per cent failed 3 plates, and 1 per cent failed 4 plates.

Defective Color Vision.—All subjects we judged on the basis of our battery of color tests to have defective red-green vision failed 5 or more

of the 18 plates, and 2 per cent failed all the plates; the number of plates failed most frequently was 15 and 16; 80 per cent failed 11 or more plates. No subjects with dichromasy in our group failed fewer than 12 plates, and subjects with anomalous trichromasy whose defect was medium or marked failed from 9 to 17 plates. Thus, error scores cannot be considered as indicating the degree or extent of color deficiency, except within rough limits.

The value of this 18 plate selection has been tested by us for two years in color vision screening in schools and in other surveys as well as by a number of ophthalmologists connected with testing for civilian aviation. It has rendered good service and so far has given satisfactory information. We feel confident in recommending this selection to meet

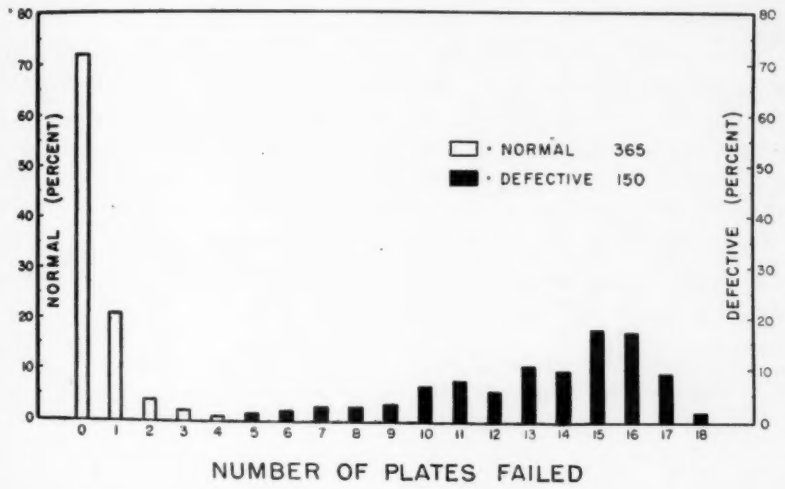
NORMAL (PERCENT)
DEFECTIVE (PERCENT)
NORMAL 365
DEFECTIVE 150
0 10 20 30 40 50 60 70 80
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18
NUMBER OF PLATES FAILED

Fig. 2.—Results for the 18 plate selection from the American Optical Company's pseudoisochromatic plates.

the present need for a test adequate to separate persons with normal from persons with defective red-green vision. This recommendation is contingent on the test being administered under the standard source of illumination.[5a]

The critical importance of illumination cannot be too strongly stressed in the use of any test designed to screen out persons with defective color vision. Merely "natural daylight" will not do. It is far too variable in quantity and quality to provide a standard test situation. For example,

5a. Since this manuscript was submitted, the American Optical Company, with the approval of the Inter-Society Color Council, has issued a revised (18 plate) edition of Pseudo-Isochromatic Plates for Testing Color Perception, which follows the suggestions and instructions included in this report.

if the amount of light is too low, the error scores of some subjects with normal color vision will lie above the critical borderline. If the light is too blue, as when reflected from north sky or from snow, again some normal subjects will have an incorrectly high error score. This is mainly because the bluish digits of the Ishihara plates (those usually seen only when red-green vision is deficient) are enhanced in coloration and are, therefore, often responded to in whole or in part by the normal subject. On the other hand, if the illumination is too yellow, as in direct sunlight or illumination by incandescent lamps, the reverse situation holds. That is, these bluish digits are rendered less pronounced, sufficiently so that many subjects whose defect in color vision is mild give the correct response and thus lower their error score sufficiently to cross the boundary line into the region of normal error score. The effect of this last type of faulty illumination has already been reported.[6]

In conclusion, we wish to repeat that the 18 plate selection is intended for screening purposes only. It is neither qualitative nor quantitative; that is, it does not classify the type of deficiency in red-green vision or, except for the extremes of error score, the degree of deficiency. It should be pointed out again that no provision is made for detecting the rare forms of defective blue-yellow vision.

Appended is a copy of the instruction sheet we have prepared for those who may be interested in utilizing this 18 plate selection as a screening test for defective red-green vision.

Instructions for Utilizing 18 of the American Optical Company's Pseudo-Isochromatic Plates for Testing Color Perception, as a Screening Test for Defective Red-Green Vision

LeGrand H. Hardy, M.D.; Gertrude Rand, Ph.D., and M. Catherine Rittler, B.A.

Source of Illumination: The Macbeth Easel Lamp. This can be purchased from Macbeth Daylighting Co., 227 W. 17th St., New York 11, New York, for $20.

Test Material: A selected series from Pseudo-Isochromatic Plates for Testing Color Perception, American Optical Co., 1940. The following plates are used: No. 25 for demonstration; Nos. 3, 4, 5, 6, 8, 9, 12, 13, 16, 17, 19, 20, 21, 23, 27, 29, 41 and 42 for testing. Other plates are to be covered with a sheet of black paper.

Instructions: The subject should be seated with his eyes about 30 inches from the test book in position on the rack of the lamp. He is shown plate no. 25 (a red *12* on a blue background, a malingering test) and instructed to read the number or numbers on this and the following charts. The examiner allows for response about 2 sec. per plate. No further instructions are to be given and no questions asked. The subject is not allowed to trace the patterns or touch the test plates.

6. Hardy, L. H.; Rand, G., and Rittler, M. C.: Effect of Quality of Illumination on the Results of the Ishihara Test, Arch. Ophth. **36**:685 (Dec.) 1946.

Responses should be immediate (within 2 sec.). Hesitant or studied responses are viewed with suspicion. Responses are to be entered on a score sheet similar to that given below. Number of plates failed is the subject's error score.

Score Sheet Name..... Date.....

Plate No.	Normal Response	Subject's Response	Plate No.	Normal Response	Subject's Response
3	56	———	17	25	———
4	27	———	19	5	———
5	8	———	20	3	———
6	6	———	21	97	———
8	42	———	23	56	———
9	56	———	27	89	———
12	57	———	29	86	———
13	86	———	41	15	———
16	9	———	42	74	———
Total errors.....					———

Interpretation of Error Score: Incorrect response to 4 or less plates indicates normal color vision; incorrect response to 5 or more plates indicates defective red-green vision.

Limitations of Test: This interpretation of error score holds only when the test is administered under the standard source of illumination. The test is for screening purposes only. It is neither qualitative nor quantitative—that is, it does not classify type of red-green defect, or the amount of defect. No provision is made for detecting defective blue-yellow vision.

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TRACHOMA

A Possible Carrier State

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TRACHOMA is one of the most widely spread diseases in the world. It may be a definite threat to the American soldier as a result of continued exposure in the Asiatic countries. It is conservatively reported that in China the disease has an incidence of about 25 per cent, implicating about 100,000,000 people.[1] In 1928 Japan was reported to have 17 per cent of its conscripts infected.[2] MacCallan[3] found there was heavy seeding of trachoma throughout the British Colonial Empire. It was especially prevalent in the Asiatic countries. The Fiji Islands are a known endemic area of trachoma. The local health authorities find it a definite epidemiologic problem. Reliable public health figures have never been available because of the lack of trained personnel to conduct an adequate survey.[4]

My interest developed during the medical examination of a group of natives employed by the United States Army stationed at Fiji. Studies were instituted to determine the incidence of trachoma among the natives and the spread of the disease to American troops.

The purpose of this paper is to present these observations and (1) to show the morbidity rate of trachoma among natives working at an American Army base in Fiji; (2) to demonstrate the presence, if any, of trachoma in troops stationed in an endemic area for long periods, and (3) to advance the hypothesis of a possible carrier state.

METHODS

Two groups of subjects were studied. One consisted of 100 natives employed at an American Army base in Fiji; the other, of 50 American soldiers stationed at the same base for a year or more. Both groups were selected at random from different parts of the camp.

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*Formerly Captain, Medical Corps, Army of the United States.

1. Miyashita, S.: Trachom in Japan und den grenzgebeiten Chinas, Internat. Cong. Ophth. **3**:169, 1929.

2. Kusama, H.: Prevalence of Trachoma and of Its Preventive Measures in Japan, Japanese M. World **8**:154 (June) 1928; cited, Bull. Hyg. **4**:264 (March) 1929.

3. MacCallan, A. F.: Trachoma in the British Colonial Empire, Brit. J. Ophth. **18**:625 (Nov.) 1934.

4. Personal communication to the author.

Clinical data were obtained from all subjects to include age, sex, general health, immediate living conditions, length of stay in Fiji, location of permanent residence, family history of ocular trouble, if obtainable, and finally, individual history of ocular disease.

All eyes were examined grossly and with the aid of a condensing lens and loupe with a 2½ magnification. The external pathologic changes in the eye were recorded on a total of 300 eyes of the 150 subjects studied. Epithelial scrapings were taken from the tarsal conjunctiva of the upper lid of both eyes of each subject. The specimens, spread thinly on glass slides, were fixed with methyl alcohol. Dilute Giemsa stain, adjusted to a p_H of 7.2, was then applied for twenty-four hours. When the specimens stained poorly, repeat smears were made until easily readable specimens were obtained. The slides were then carefully examined for Prowazek-Halberstädter inclusions at a magnification of 950.

Fifteen patients were followed at monthly intervals over a four month period for beginning external ocular disease. One patient was examined in the same manner at weekly intervals over this period. In this case epithelial scrapings were studied for inclusion bodies after each examination.

Personal Data and Histories.—Native Subjects: The ages of this group ranged from 14 to 60 years, with a mean average of 24.8 years. Twelve of these subjects were females. All stated that they were in good general health. They attended their jobs at the post as laborers with regularity. The great majority lived in small wooden shacks or thatched huts. Ordinary hygienic measures, such as toilet facilities, use of individual towels and fly control, were practically nonexistent. The fact that practically all the subjects harbored a museum variety of intestinal parasites attested to the lack of sanitation among these people. No natives lived within the Army base confines. All were born in Fiji. In addition to the aboriginal Melanesians of this group, there were East Indians, Melanesian-Caucasian half-castes and Rotumans.[5] Because of language difficulties and low educational standards, family histories were considered entirely unreliable. For this reason, they were excluded from this report.

Twenty-five of the native subjects stated that they had had symptoms suggesting inflammation of the conjunctiva. Among these complaints were irritation, redness, photophobia and discharge. Six of these subjects complained of some of these symptoms at the time of examination. The duration of symptoms in the entire group varied from two days to two years. None of this group had received any specific treatment.

American Subjects: The average age of this group was 27.8 years, with a range of 19 to 47 years. All were men and in robust health. Their homes were located throughout the United States, with no locality predominating. Strictly enforced military sanitation prevailed at their

5. Rotumans are a Polynesian-Chinese admixture derived from Rotuma, an island on the periphery of the Fiji group.

garrison station in Fiji. All had been at this base for from twelve to thirty-seven months, with an average stay of 24.8 months. None knew of any chronic inflammatory disease of the eyes in their families. All stated that they had never had trachoma or any condition suggestive of it.

Military regulation prohibits our men from consorting with the natives in this area. In general this ruling was complied with.

Clinical Ocular Findings.—Native Subjects: Twenty-two of the 100 natives, or 22 per cent, showed evidence of active trachoma. This consisted of redness, thickening and definite scarring or follicle formation of the tarsal conjunctiva, together with infiltration and vasculariza-

TABLE 1.—*Summary of Clinical Data on Patients Showing Evidence of Trachoma*

Case No.	Age, Yr.	Duration of Symptoms	Conjunctival Changes: Thickening *	Scarring †	Hyperemia ‡	Follicles §	Pannus ¶	Entropion, Trichiasis
7	48	2 yr.	+	+	+	—	+	—
10	30	2 yr.	++	++	++	—	++	—
12	34	2 yr.	++	++	++	—	++	—
13	19	None	+	+	++	—	+	—
17	19	None	++	++	++	—	+	—
18	39	3 wk.	++	+	+	—	+	—
30	23	2 yr.	+++	++	+++	—	+	—
52	14	None	+	+	++	—	+	—
53	21	None	+	++	+	—	+	—
55	43	2 yr.	+	++	+	—	+	—
57	22	None	+	+	+	—	++	—
60	18	None	+	—	+	+	+	—
61	35	1 wk.	+	+++	+	—	++	Entropion
62	28	1 yr.	+	++	++	—	++	—
82	30	2 wk.	—	+	+	—	+	—
88	38	None	+	+	+	—	+	—
91	42	1 wk.	+++	+++	+++	—	+++	Entropion, trichiasis
92	20	None	+	++	+	—	+	—
93	18	None	++	+	++	+	+	—
98	41	None	+	+	+	—	+	—
99	60	2 wk.	++	++	++	—	++	Entropion
100	28	2 wk.	+	+	+	—	+	—

* Barely discernible heaping is indicated by +; uniform, early discernible thickening by ++, and marked thickening, with the closed lid overly convex, by +++.

† Scarring which is barely discernible and may require the aid of a loupe is indicated by +; scarring easily discernible grossly, by ++, and gross scarring, causing malformation of the lid, by +++.

‡ A mild, rose-colored blush is indicated by +; a bright red conjunctiva but discrete vessels discernible, by ++, and a bright, beefy red conjunctiva, with individaul vessels not made out, by +++.

§ The absence of conjunctival follicles is indicated by —; a few scattered follicles are indicated by +.

¶ Vessels 2 to 3 mm. in from the limbus are indicated by +; vessels 3 to 6 mm. in from the limbus, by ++, and vessels more than 6 mm. in from the limbus, by +++.

tion of the cornea. Each of the native patients with positive clinical evidence of trachoma had practically identical lesions in the two eyes. Most of these persons had trachoma stage III (table 1).

Twenty-six of the remaining 78 trachoma-free subjects showed conjunctival hyperemia. This was mild and bilateral in all patients. Particularly close scrutiny revealed no evidence of trachoma. Except for occasional pingueculae, pterygiums and the like, the eyes of the

remaining 52 persons seemed entirely normal on external examination. Six patients gave a history of ocular symptoms for six months or more. Five of these (83.3 per cent) had clinical evidences of trachoma. Seventy-five subjects had no symptoms referable to the eyes; yet 10 of these (13.3 per cent) had clinical trachoma (table 2).

American Subjects: None of the American soldiers had clinical trachoma. On the other hand, all but 6 of the men showed mild conjunctival hyperemia. The condition was always bilateral and was noted only on the palpebral portions. Occasional pingueculae and pterygiums were also noted.

TABLE 2.—*Relation of History of Ocular Inflammation to Clinical Evidence of Trachoma*

Length of History of Inflammation	Subjects with Clinical Trachoma	Trachoma-Free Subjects	Total No. of Subjects	Percentage with Clinical Trachoma
More than 6 months	5	1	6	83.3
1 to 6 months	1	2	3	33.3
Less than 1 month	6	10	16	37.5
None	10	65	75	13.3
Total number	22	78	100	22.0

TABLE 3.—*Distribution of Inclusion Bodies in One or Both Eyes in Patients with Microscopically Suggestive Evidence of Trachoma*

	Unilateral Inclusion Bodies		Bilateral Inclusion Bodies		Total	
	No. of Eyes	No. of Patients	No. of Eyes	No. of Patients	No. of Eyes	No. of Patients
Patients with trachoma	2	2	26	13	28	15
Patients free of trachoma	15	15	24	12	39	27
Total number	17	17	50	25	67	42

Laboratory Findings.—Native Subjects: For this study the natives fell into two convenient groups—those with trachoma and those who were free from trachoma. Fifteen of the 22 patients with clinical trachoma also showed Prowazek-Halberstädter inclusion bodies. Thirteen of these 15 patients showed these bodies in smears of material from both eyes. Two of the 15 patients exhibited these bodies in only one eye. Fifteen of 27 patients who were free of trachoma clinically showed the bodies in smears from one eye only, and 12 showed inclusions in smears from both eyes (table 3). Of the 22 trachomatous subjects, 68.2 per cent then, revealed inclusion bodies.

Of the remaining 78 subjects, 27, or 24.6 per cent, harbored inclusion bodies in one or both eyes. It is to be remembered that none of these 78 subjects had shown the least evidence of the disease. These

inclusion bodies were microscopically indistinguishable from those observed in smears from the trachomatous patients (figs. 1 and 2).

When the two groups are considered together, one finds that of 100 natives, 42 exhibited Prowazek-Halberstädter inclusion bodies of the conjunctival epithelium (table 4).

Fig. 1.—Photomicrograph (1,500 magnification) of conjunctival scrapings from the eye of a Fijian native with active trachoma. Arrows indicate inclusions in the epithelial cells in various phases of development.

American Soldiers: Prolonged and careful search revealed no inclusion bodies of any description. Rarely a few isolated bacteria were noted. These showed no uniformity as to staining properties or morphologic characteristics.

Follow-up Observations.—Fifteen of the 27 natives who harbored inclusion bodies and yet had no clinical disease were examined at monthly intervals. Signs of early cicatrization, follicle formation and corneal vascularization were searched for. These were persistently absent over a four months' period of observation.

Fig. 2.—Photomicrograph (1,500 magnification) of conjunctival scrapings from the eye of a Fijian native who showed no clinical evidence of trachoma, either past or present. Arrows indicate inclusion bodies in various phases of development. In no way were these inclusion bodies distinguishable from those observed in cases of clinical trachoma.

One native who showed the same changes as the natives in the preceding group was followed at weekly intervals for four months in

the same way. Smears for inclusion bodies were examined at the same time. Although the smears showed persistently heavy seeding, no clinical evidence of beginning trachoma could ever be detected.

COMMENT

Clinical Trachoma in Fiji.—It was noted that 22 per cent of 100 natives working at an Army base in Fiji had clinical evidence of trachoma. Although these natives had come from widely separated areas of the islands, they did not represent a completely random sample of the population. The fact that they were able to be employed as laborers at an Army installation of limited size indicated that they were in good general health, fell into a definite age group, were not industrially blind and constituted a small sampling of the population. The disparity in numbers of males to females in this study was also out of keeping with the general population. In spite of these shortcom-

TABLE 4.—*Number of Patients Showing Prowazek-Halberstädter Inclusion Bodies in Clinically Trachomatous and Nontrachomatous States*

Type of Patient	Number of Patients Showing Inclusion Bodies	Number of Patients Showing No Inclusion Bodies	Total Number of Patients	Percentage of All Patients Showing Inclusion Bodies
Trachomatous	15	7	22	68.2
Free from trachoma	27	51	78	34.6
Total	42	58	100	42.0

ings, it is believed that these findings give a suggestion of the general picture of trachoma in Fiji. Extensive and thoroughgoing surveys are still awaited for this area.

The incidence of trachoma of 22 per cent found in this study is of particular interest as compared with the consolidated statistics on American Indians made in 1938, which showed a rate of 20.2 per cent.[6] Since that year the sulfonamide drugs have decreased that morbidity rate remarkably.[6]

None of the subjects in the present series had received specific therapy for trachoma prior to this study. Their primitive, agricultural mode of living has changed little in the past decade.

Trachoma Among American Troops.—On the basis of correspondence with a number of oculists on military service overseas, Thygeson [8] expressed the opinion that trachoma does not represent a serious

6. Forster, W. G., and McGibony, J. R.: Trachoma, Am. J. Ophth. **27**:1107 (Oct., pt. 1) 1944.

7. Footnote deleted by the author.

8. Thygeson, P.: Personal communication to the author, April 1945.

military problem. My findings would tend to bear out the validity of this opinion.

Although a slit lamp was not available, I am in agreement with MacCallan's [9] statement that careful observation with a condensing lens and corneal loupe will disclose pannus formation. By this means early changes were made out at the limbus in trachomatous patients. These included distortion of the capillary loops, early vascularization beyond the limbus and corneal infiltrations. Our subjects who were free from trachoma showed none of these changes.

The high prevalence of conjunctival hyperemia among our troops has caused much speculation. It was seen in almost all American troops stationed in Fiji for over a year. Smears and bacterial cultures have failed uniformly to show any specific causative organism. Several other Army oculists stationed here found the same condition among their patients. Over a three year period none had ever found anything that resembled trachoma among these soldiers. Thousands of troops have been seen during this time in the various Army eye clinics in Fiji. Tropical sunlight, heavy exposure to dust and the prevalence of plant allergens may account for some of the conjunctival hyperemia.

The absence of inclusion bodies in the conjunctival scrapings would seem to confirm our clinical findings in regard to trachoma among our men.

The "Carrier State."—Recent papers by several of the leading world authorities on trachoma have made no mention of trachoma carriers.[10] In the present series of cases, 27 of 78 subjects (34.6 per cent) who were free from trachoma had demonstrable conjunctival inclusion bodies. These bodies were indistinguishable from those seen in each of the cases of trachoma.

Two typical slides showing these inclusion bodies from the trachoma-free subjects were sent to the section on virology of the Eighteenth Army medical general laboratory. The staff reported that the smears were consistent with those seen in cases of trachoma.

From all available sources (civilian as well as military) there is excellent reason to believe that inclusion blennorrhea, lymphogranuloma

9. MacCallan, A. F.: Clinical Signs of Trachoma, Rev. internat. du trachome **11**:3 (Jan.) 1934.

10. (*a*) MacCallan.[8] (*b*) Forster and McGibony.[6] (*c*) MacCallan.[9] (*d*) Julianelle, L. A., and Smith, J. E.: A Statistical Analysis of Clinical Trachoma, Am. J. Ophth. **26**:158 (Feb.) 1943. (*e*) Thygeson, P.: Viruses and Virus Diseases of the Eye, Arch. Ophth. **29**:285 (Feb.); 488 (March); 635 (April) 1943; (*f*) Trachoma, in Piersol, G. M.; Bortz, E. L., and others: Cyclopedia of Medicine, Philadelphia, F. A. Davis Company, 1935. (*g*) Lindner, K., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936.

venereum and psittacosis do not exist in this locality. The inclusion bodies of these diseases seem to be the only ones to be mistaken for trachoma bodies in the eye.[10e] The absence of these diseases and the prevalence of trachoma in this area support the opinion that the inclusions noted are derived from trachoma.

There is good reason to believe that the microscopic inclusions are Prowazek-Halberstädter corpuscles. The only other objects with which they could be confused are pigment granules or technical artefacts. We are convinced that they were neither. The usual conjunctival pigment, when present, consists of melanin. The tarsal conjunctiva is an unusual site for this pigment, and melanin was never seen grossly here in our patients. Microscopically, conjunctival melanin is seen as diffusely scattered, intracellular greenish granules (Giemsa stain), which do not form conglomerate masses similar to those of inclusion bodies and do not invade the nuclei of epithelial cells. The bodies we observed were always derived from the tarsal conjunctiva, tended to form characteristic morphologic groups, took a fairly pure blue stain (Giemsa) and occasionally were seen in the nuclei of the conjunctival cells. The morphologic conglomerations, such as "cape" formations and initial body groups, were identical with those described by Lindner and Thygeson in their various excellent articles on the subject. Extracellular elementary bodies were also noted in some instances.

From what has been said regarding the characteristics of the inclusions noted, I feel strongly that they were not technical artefacts. I examined every slide in this study. When, on occasion, a reasonable doubt existed, the slide was called negative for Prowazek-Halberstädter bodies. The fact that the virologists at the Eighteenth Army medical laboratory concurred in our belief that the inclusion bodies were consistent with those observed in cases of trachoma strengthens our feeling on the matter. The slides which they reviewed were taken from persons free of clinical ocular disease. Furthermore, all the slides in this study were treated with the same technic. The natives' slides showed many inclusion bodies, but not one of the Americans' did. This indicates, we feel, that the bodies noted were not technical artefacts.

In order to rule out a possible preclinical or incubation phase of trachoma, a follow-up study was instituted. Fifteen persons who were free from trachoma and who showed inclusion bodies were examined at monthly intervals for signs of beginning trachoma. Over a period of four months I was unable to detect the least evidence of scarring, follicle formation or corneal involvement. One subject was examined for inclusion bodies, as well as clinical signs, at weekly periods. Although his smears were repeatedly positive, no clinical

changes could be detected in a four month survey. Since the incubation period for trachoma is five to twelve days, we felt that these patients did not represent an incubation phase of the disease. The findings in these patients are consistent with a carrier state in trachoma and can be fully explained on this basis.

Broadhurst and associates [11] demonstrated a carrier state in patients with virus infection of the throat. Bodies of the same type which were found in the throat were also found in the eyes of 2 subjects. Carriers of the virus of other diseases, such as herpes, have been known to be asymptomatic over a period of years.[12] Trachoma is generally accepted to be a virus disease. My observations with respect to a carrier state in trachoma conform with those of workers on carrier states in other virus diseases.

It is interesting to speculate on the origin of the carrier state in our cases. MacCallan and others reported that trachoma typically begins imperceptibly and may go on to spontaneous cure.[13] It may well be that our "carriers" represent instances of just such a state.

Without a virology laboratory or suitable experimental animals at my disposal, I have been unable to demonstrate the transmissibility or pathogenicity of the bodies in these "carriers." Until this is done, a true carrier state in trachoma will not be fully proved. It is hoped that these important studies will soon be under way.

SUMMARY AND CONCLUSIONS

Of 100 natives working at an American Army base in Fiji, 22, or 22 per cent, had clinical evidence of active trachoma.

Typical Prowazek-Halberstädter inclusion bodies were seen in the conjunctival epithelial scrapings of 15 of these patients, an incidence of 68.2 per cent.

Evidence is presented which suggests the existence of a carrier state in trachoma. Seventy-eight native subjects employed by the American Army showed no evidence of clinical trachoma. Of these, 27, or 34.6 per cent, had inclusion bodies in the conjunctival epithelial scrapings morphologically indistinguishable from those observed in frankly trachomatous patients. Transmissibility and pathogenicity of the virus of trachoma from these patients still await demonstration.

11. Broadhurst, J.; Liming, R.; MacLean, E., and Taylor, I.: Cytoplasmic Inclusion Bodies in Human Throat, J. Infect. Dis. **58**:134 (March-April) 1936.

12. Broadhurst, J.; MacLean, E., and Taylor, I.: Increased Incidence of Cytoplasmic Virus Bodies in Human Throats in New York City Area, J. Infect. Dis. **73**:195 (Nov.-Dec.) 1943.

13. MacCallan, A. F.: Trachomatous Conjunctivitis: Its Surgery and Pathology (Hunterian Lecture), Lancet **1**:215 (Jan. 25) 1936.

Of 100 native subjects studied, both those with and those without clinical trachoma, 42, or 42 per cent, harbored Prowazek-Halberstädter inclusions in the conjunctival epithelium.

One hundred eyes of 50 American soldiers who have been based in an area endemic for trachoma (Fiji) for one to three years were examined for evidence of the disease. Clinical and laboratory evidence indicated the absence of trachomatous infection among these men.

With proper sanitation and segregation of the soldier from the native population, trachoma would not seem to present a military problem.

Technician Fourth Grade Hugh P. Earle, Medical Department, Army of the United States, prepared the laboratory material used in this report.

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TREATMENT OF LINDAU'S DISEASE

Report of a Case

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LINDAU'S disease is an angiomatosis of the central nervous system characterized by the occurrence of single or multiple hemangioblastomas in the cerebellum, brain stem or spinal cord, associated with angiomatosis of the retina (von Hippel's disease). In addition, there are present various congenital lesions of other organs, such as cystic disease of the kidneys and pancreas. Lindau found that the retinal lesions made their appearance at the average age of 25, while cerebellar symptoms did not become manifest until about fourteen years later, when partial blindness had usually set in. Cox and Trumble [1] pointed out that autopsy of patients with this disease has frequently revealed a hemangioma of the spinal cord which clinically had shown no evidence of its presence. The case to be described is unique in that compression of the cord by a vascular tumor was the primary and outstanding feature and only later was a typical, nonsymptomatic retinal angioma discovered on routine ophthalmoscopic examination.

As in the case of many other tumors, a controversy exists in the treatment of von Hippel's disease, some preferring electrosurgical measures and others irradiation. The successful destruction of retinal angiomas by diathermy puncture was first described by Weve,[2] of the

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1. Cox, L. B., and Trumble, H. C.: Tumors and Malformations of Blood Vessels of Brain and Spinal Cord, M. J. Australia **2**:308-319 (Aug. 26) 1939.

2. Weve, H.: Bowman Lecture: On Diathermy in Ophthalmic Practice, Tr. Ophth. Soc. U. Kingdom **59**:43-80, 1939.

Netherlands, in 1939 and was first performed in this country by Lewis [3] in 1942. Guyton and McGovern,[4] in 1943, obliterated multiple angiomatous lesions of the retina, which had been progressing in spite of roentgen irradiations, with 1,500 r to each eye. In view of these favorable reports, and because the retinal angioma, in an early stage, was situated near the equator, readily accessible to diathermy puncture, we employed this method of treatment.

REPORT OF CASE

R. C. J., a Negro soldier aged 27, in November 1943 noted the gradual onset of weakness of the right leg with dragging of the toes of the right foot and some difficulty in starting the urinary stream. During the next month he complained of shooting pains up and down the spine, and the weakness of his right leg progressed so that he became unable to climb stairs. Examination revealed spasticity of the right leg in walking. The deep reflexes of the right upper extremity and of both lower extremities were hyperactive, with bilateral ankle clonus and absence of the abdominal and cremasteric reflexes. The plantar response, which was elicited bilaterally, consisted in fanning of the smaller toes without dorsiflexion of the great toes. There was pronounced weakness of the right lower extremity with some atrophy of the muscles of both the thigh and the calf and a conspicuous increase in their tone. The left leg was only slightly weak and spastic, with no atrophy. A band of hypalgesia was present over the ninth and tenth dorsal dermatomes on the right side, with minimal hypalgesia of the left leg to about the level of the thigh. Spinal puncture revealed a positive reaction to the Queckenstedt test and a total protein content of 800 mg. per hundred cubic centimeters. A roentgenogram of the spinal column showed no evidence of a pathologic process, but a myelogram taken with "pantopaque" (a mixture of ethyl esters of isomeric iodophenyl undecylic acids) showed a filling defect from the eighth to the eleventh dorsal segment and a slight defect as high as the first dorsal segment. Routine laboratory tests, including blood counts, urinalysis and a Wassermann test of the blood, revealed nothing abnormal.

Although the presence of hyperactive reflexes in the right upper extremity could not be explained, the patient was thought to have a tumor of the cord, and on Feb. 26, 1945 a laminectomy was done from the ninth dorsal to the first lumbar vertebra. A graphic description by Major G. L. Maltby of the observations at operation follows.

"On exposure of the cord, an interesting and amazing picture came into view. The dorsum of the cord was almost completely covered with numerous vermiform, dilated blood vessels. On the right side of the posterior portion of the cord, at a point about the level of the eleventh dorsal vertebra, there was a pinkish, flesh-colored tumor nodule, about ¼ inch (6 mm.) in diameter. Several of the vessels seemed to enter this tumor mass and others to pass around it. At the upper end of the dural opening a great mass of dilated veins seemed to become more profuse and resembled tightly coiled ringlets of curled hairs."

Excision of this great network of vessels was deemed inadvisable, and a biopsy specimen was taken from the small tumor nodule. Major Webb Haymaker, of the Army Institute of Pathology, gave the following description of the specimen (fig. 1):

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3. Lewis, P. M.: Angiomatosis Retinae: Successful Treatment (Puncture Diathermy) in One Case, Arch. Ophth. **30**:350-354 (Aug.) 1943.

4. Guyton, J. S., and McGovern, F. H.: Diathermy Coagulation in Treatment of Angiomatosis Retinae: Case, Am. J. Ophth. **26**:675-684 (July) 1943.

"The specimen consists of only a few small fragments of tissue. The tumor is composed of a number of small blood spaces, lined with endothelium and supported by polymorphous cells with highly chromatic, spherical, oval or irregularly elongated nuclei and abundant, homogeneous cytoplasm with ill defined borders. Within the

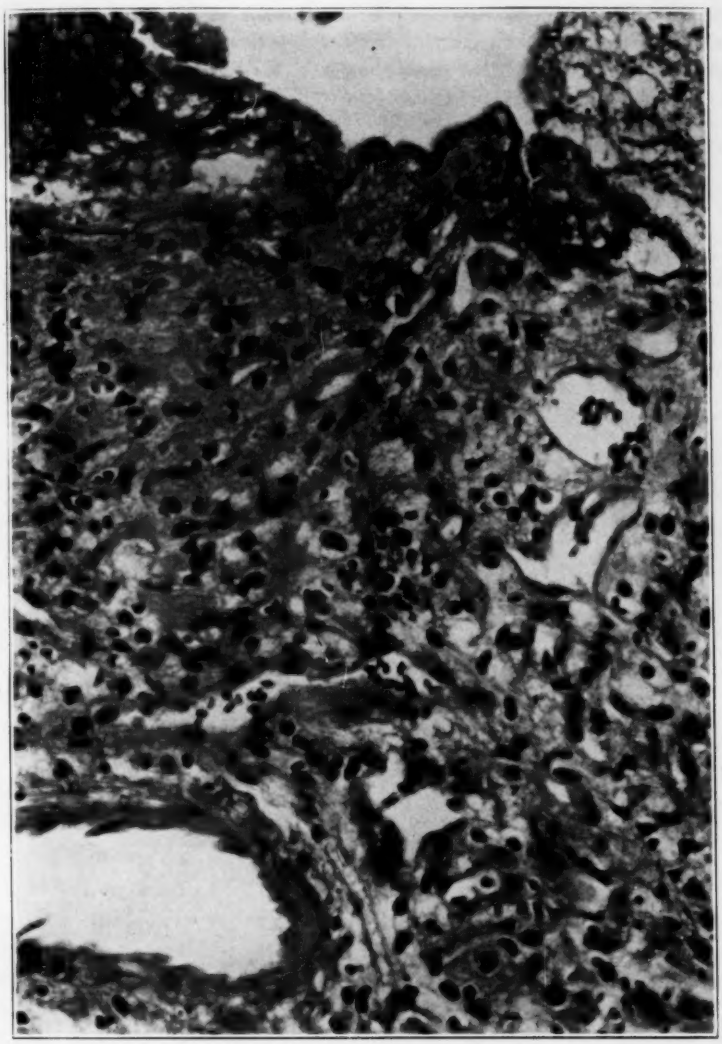

Fig. 1.—Photomicrograph of the tumor of the spinal cord. United States Army Medical Museum, neg. no. 95103; × 400.

tumor proper there are also a few large vessels having the appearance of veins. The adjacent portion of the spinal cord is entirely necrotic and is being invaded *en masse* by the tumor. The appearance of the tumor conforms to that of a sclerosing hemangioma."

After roentgen therapy (5,400 r to the lumbodorsal portion of the spine), the patient was discharged from the Army to a veterans administration hospital, on June 19, 1945. Examination here revealed hyperreflexia of the upper extremities and persistence of the spastic paraplegia, although the patient stated that his hands were stronger and the control of urine better. There was no family history of blindness or disease of the central nervous system. Routine ophthalmologic examination revealed vision of 20/20 in each eye, and the patient had no ocular complaints. The right eye was normal, but in the fundus of the left eye the superior temporal artery and vein were greatly dilated and tortuous, with beading of the artery.

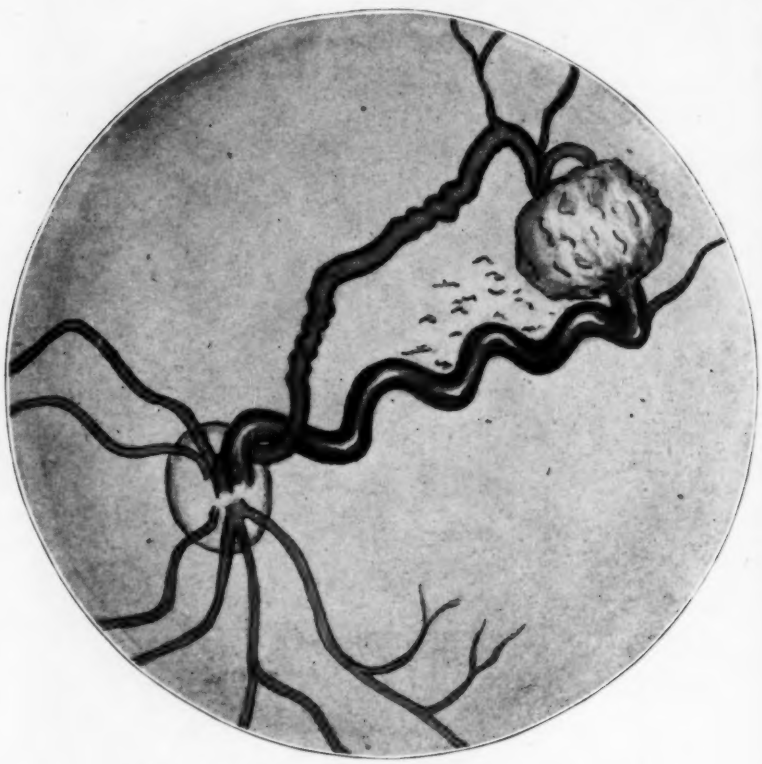

Fig. 2.—Appearance of the fundus of the left eye.

When the pupils were dilated with 5 per cent solution of encatropine ("euphthalmine") hydrochloride, these vessels could be seen to end far in the periphery of the retina near the ora serrata, on either side of an almost circular reddish striated mass, measuring 1.5 disk diameters. Below this slightly elevated mass were a few horizontal yellow streaks of exudate in the retina. The macula was normal (fig. 2).

Operation and Course.—Since von Hippel's disease ultimately leads to blindness, electrocoagulation of the retinal angioma was performed on August 6, with retrobulbar anesthesia induced with 1 per cent procaine hydrochloride. A conjunctival flap was made superiorly, and the upper half of the globe was exposed by dividing the tendon of the superior rectus muscle. The Walker diathermy apparatus

was used, a single needle, consisting of a fine stylet wire guarded with a piece of glass capillary tubing in the plastic holder, being employed. A trial puncture of the sclera was made with the diathermy needle at the approximate position of the angioma. The small white lesion so produced in the retina was readily visible with the ophthalmoscope ⅓ disk diameter above and nasal to the angioma. With this spot as a guiding point, ten punctures were made into the tumor, the coagulated portions turning white. After this procedure only the upper surface of the lower quarter of the angioma remained pink. The bubbles of vitreous which formed elevated the two large vessels arising from the tumor and thus prevented their destruction. There was no hemorrhage, and the conjunctiva was closed after resuturing the severed rectus muscle. The postoperative reaction in the eye was mild. On the third day the untreated eye was uncovered, and on the seventh day both eyes were uncovered and the patient allowed out of bed in a wheel chair. The left eye was atropinized for three weeks. A month later severe acute pyelonephritis developed, which was controlled only with a cystotomy and intensive treatment with sulfadiazine and penicillin. The patient has been bedridden since, with recurrent exacerbations of pyelonephritis.

Examination on June 6, 1946 revealed no abnormality in the right eye. In the left eye the superior temporal vein had shrunk to the same caliber as the inferior temporal vein. Near the site of the angioma it became hairlike and almost invisible. The superior temporal artery was slightly larger than the inferior temporal artery and was still beaded. The upper half of the lesion was replaced with white scar tissue; the lower half was still pink but was no longer striated or elevated, and the yellow streaks in the retina near it were absent. Visual acuity in each eye was unchanged. Neurologic examination revealed an increase in the spastic paraplegia with incontinence of feces. Both upper extremities were weak, with hyperactive reflexes. The right arm was weaker than the left and gave a positive Hoffmann response.

COMMENT

In their pathologic study of vascular lesions of the spinal cord, Turner and Kernohan (1941)[5] classified hemangioma and hemangioblastoma as vascular neoplasms and stated that the difference between them was one of degree, "the latter appearing as a more cellular and microscopically more active tumor than the former." They added that these vascular tumors are rarely architecturally pure and that intermediate forms are frequently seen. Bailey and Ford (1942)[6] used the terms hemangioma and hemangioblastoma interchangeably, pointing out that the former term is the one more often employed by general pathologists for vascular tumors of the skin and other viscera, whereas the latter term is used particularly by neuropathologists to apply to vascular tumors of the brain. Since the lesion of angiomatosis retinae is a hemangioblastoma of the cerebral part of the retina, it is inconceivable that a similarly rare vascular tumor elsewhere in the central

5. Turner, O. A., and Kernohan, J. W.: Vascular Malformations and Vascular Tumors Involving the Spinal Cord, Arch. Neurol. & Psychiat. **46**:444-463 (Sept.) 1941.

6. Bailey, O. T., and Ford, R.: Sclerosing Hemangiomas of the Central Nervous System, Am. J. Path. **18**:1-27 (Jan.) 1942.

nervous system would have a different ultimate histologic structure. Indeed, we should feel almost justified in making the diagnosis of angioblastoma of the cord on the basis of the excellent description of the tumor as seen at operation, which so closely resembles the description and photographs of the angioblastoma of the spinal cord seen at necropsy in the case of Lindau's disease described by Davison, Brock and Dyke.[7]

It is impossible to make a diagnosis of the complete Lindau pathologic complex in this case until autopsy is performed, for only then can the other congenital visceral anomalies be disclosed. Craig, Wagener and Kernohan [8] expressed the opinion, however, that one is justified in making a diagnosis of Lindau's disease on finding an angiomatosis retinae together with a surgically proved cerebellar hemangioblastoma. This view is generally accepted. Similarly, we feel that the diagnosis of Lindau's disease is established in our case by the presence of von Hippel's disease in association with a surgically verified hemangioma (hemangioblastoma) of the spinal cord. In a review of the literature through June 1939, MacDonald [9] found only 10 cases of Lindau's disease in which the diagnosis had been verified by operation during life. MacNab,[10] in 1941, reported another case, and in the same year Craig and associates [8] reported 4 more. Our case can now be added to this list and is the first to be verified by laminectomy and the observation of a vascular tumor of the spinal cord. Since the aim of every surgeon is to make a preoperative pathologic diagnosis, it is accordingly recommended that the periphery of the fundus be examined with mydriasis in all cases of suspected tumor of the cord, just as Cushing and Bailey [11] advised in all cases of suspected cerebellar tumor.

We wish to emphasize the ease and safety of the electrocoagulation procedure and the fact that it is apparently unnecessary to destroy the feeding vessels of the tumor. While a pink lesion remains at the original site in the present case, we believe that the retinal angioblastoma has been occluded, for there is no longer any retinal exudate (a tumor product) and the pink area is flat and nonstriated. Moreover, the vessels of the tumor have shrunken to almost normal size, with partial

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7. Davison, C.; Brock, S., and Dyke, C. G.: Retinal and Central Nervous Hemangioblastomatosis with Visceral Changes, Bull. Neurol. Inst. New York **5**:72-93 (Aug.) 1936.

8. Craig, W. M.; Wagener, H. P., and Kernohan, J. M.: Lindau-von Hippel Disease, Arch. Neurol. & Psychiat. **46**:36-54 (July) 1941.

9. MacDonald, A. E.: Lindau's Disease: Report of Six Cases with Surgical Verification in Four Living Patients, Arch. Ophth. **23**:564-576 (March) 1940.

10. MacNab, G. H.: Lindau's Disease: Case Report, Proc. Roy. Soc. Med. **34**:324-325 (April) 1941.

11. Cushing, H., and Bailey, P.: Tumors Arising from the Blood Vessels of the Brain, Springfield, Ill., Charles C Thomas, Publisher, 1928.

obliteration of the vein. Unfortunately, in spite of the roentgen therapy the involvement of the spinal cord is progressing inexorably. It is to be noted that radiation was given only to the lumbodorsal area, while the neurologic signs point to invasion of the brain stem or upper cervical part of the cord by the neoplasm or by a concomitant syringomyelic cavity, as reported by König and Schoen.[12] Hirschfeld,[13] in 1944, achieved a favorable result in the roentgen treatment of a patient with Lindau's disease presumably involving the medulla. He observed that there were no reports in the literature of any attempt to treat spinal or medullary hemangioblastoma with roentgen radiation, an indication chiefly of the rarity of the lesion. It is planned to give our patient radiation to the upper cervical portion of the cord in the near future.

SUMMARY

A case of Lindau's disease with unusual manifestations is described. Compression of the cord was produced by a histologically verified sclerosing hemangioma arising from the cord, whereas a solitary coexisting retinal angioma was asymptomatic. The retinal tumor was attacked directly by electrocoagulation without any loss of vision, and the success attained by this means furnishes additional support for the view that puncture diathermy is the method of choice in the treatment of von Hippel's disease. The tumor of the cord had previously been treated with roentgen radiation, but one year later further involvement of the cord had occurred. It is suggested that fundoscopic examination with use of a mydriatic be made preoperatively in all cases of suspected tumor of the cord to detect isolated cases of Lindau's disease.

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12. König, E., and Schoen, H.: Diffuse Angiomatosis of Medulla and Spinal Cord with Syringomyelia (Lindau Syndrome), Beitr. z. klin. Chir. **170**:239-265, 1939.

13. Hirschfeld, M. H.: Hemangioblastoma of Medulla: Lindau's Disease; Response to Radiation Therapy, J. Nerv. & Ment. Dis. **99**:656-659 (May) 1944.

CORNEAL LIGHT REFLEX IN DIAGNOSIS OF RETINAL CORRESPONDENCE

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BROOKLYN

EYES normally work together in close harmony by virtue of an intimate understanding between the fovea or macula of one eye with that of the other. These retinal components are called corresponding retinal points, and when identical impulses strike both maculas they register a fusion response in the brain which is called bimacular, or binocular, fixation (fig. 1). It is quite possible that such identical impulses may strike these corresponding retinal points and not register

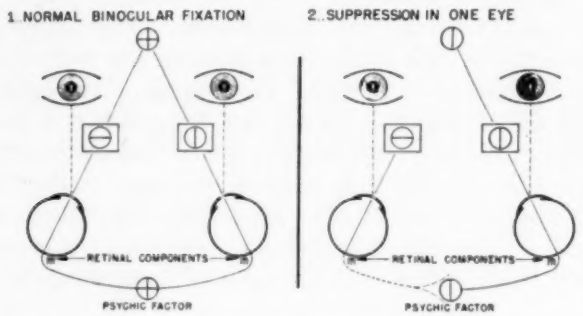

Fig. 1.—Corresponding retinal points.

fusion because of a break in some part of the visual pathway behind either eye, so-called monocular suppression. Therefore, the psychic assimilation or fusion of impulses on corresponding retinal points determines whether or not the person has retinal correspondence.[1]

RETINAL CORRESPONDENCE

Any form of retinal correspondence, whether it is normal or abnormal, is associated with some form of psychic interpretation, in contradistinction to so-called corresponding retinal points, which have a purely geometric conception. The term, therefore, is inadequate because

Read in part at a meeting of the New York Society of Clinical Ophthalmology, Oct. 7, 1946.

1. Bielschowsky, A.: Etiology of Strabismus, Am. J. Ophth. **20**:478-489, 1937; Physiology of Ocular Movements, ibid. **21**:843-854, 1938.

it stresses the retinal element only. A more correct term would probably be psychoretinal correspondence. The terms retinal correspondence and retinal projection are used interchangeably; the former suggests a sensory component and the latter a motor component. Here, too, such refinements in terminology appear unnecessary, and it is the central, or psychic, factor which determines the ultimate response. The term retinal projection would seem to place the retina in the same category as automobile headlights or reflectors.

Normal retinal correspondence is a term employed to indicate registration of identical impulses on corresponding retinal points and assimilation in the brain of a fused image. The term itself does not necessarily indicate that the eyes are operating in normal fashion. It merely indicates that the brain can be made to fuse images that strike both maculas. In normal binocular fixation such correspondence is taken for granted. However, in a case of manifest squint it is conceiv-

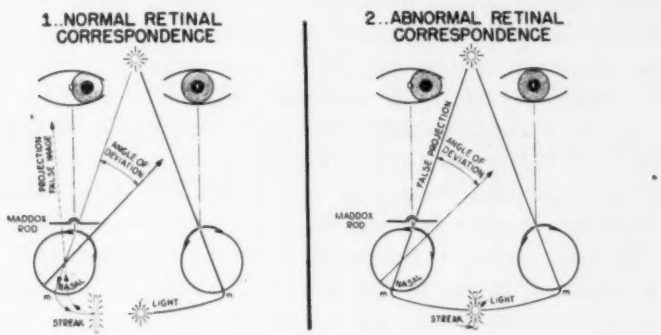

Fig. 2.—Retinal correspondence and false projection.

able that a common light source will not reach both maculas directly, and, therefore, it is necessary through artificial means to shift or bend the light rays so that they will reach the two maculas simultaneously and thereby register fusion. Because normal retinal correspondence means bimacular fusion, it is important to indicate whether it is part of normal binocular fixation or part of a manifest squint.

Abnormal retinal correspondence,[2] in contradistinction to normal retinal correspondence, is a macular-paramacular association because the deviating eye learns to accept a paramacular stimulus. As such, it is practically always associated with a manifest squint.

FALSE PROJECTION

False projection is a term often used to indicate the projection of a false image in case of binocular diplopia associated with paralytic

2. Travers, T. a'B.: Origin of Abnormal Retinal Correspondence, Brit. J. Ophth. **24**:58-64, 1940.

squint (fig. 2). It is also used loosely and synonymously with abnormal or anomalous or secondary correspondence or projection. Binocular diplopia is neither normal nor abnormal retinal correspondence, for there is no psychic adjustment or correspondence. It is in reality a disturbance in normal retinal correspondence in which normally corresponding retinal points have been thrown out of alinement.

In abnormal retinal correspondence the deviating eye develops a new angle of projection which corresponds to or approximates the primary, or binocular, angle. This angle is also called "false projection." While there is no uniform terminology, it is important to specify whether or not this false projection is part of a readjusted abnormal retinal correspondence or of a paralytic squint or of normal retinal correspondence.

EVOLUTION OF ABNORMAL RETINAL CORRESPONDENCE

Any speculation on the origin of abnormal retinal correspondence is merely hypothetic because a manifest squint may show normal or abnormal retinal correspondence or a combination of the two. However, some form of visual understanding might help one to explain the nature of fusion response associated with this condition. One can conceive of a disturbing diplopia with paralytic squint becoming gradually less with time because of suppression of a false image. Instead of binocular diplopia, one now has monocular vision with concomitant relative suppression in the nonfixing eye. Spontaneous fusion is for all practical purposes absent because fixation is monocular. It is, however, suppressed only as a result of such deviation, and artificial stimulation of both eyes can often be shown to awaken momentary or temporary return of the binocular diplopia corresponding to the amount of the deviation and the position of the displaced corneal light reflex. This would indicate normal retinal correspondence.

In another case of squint, however, with the same amount of deviation such artificial light stimulation of the eyes will produce a diplopia with the two images close together even though the position of the eye and the displacement of the corneal light reflex suggest much greater separation of false and true images. This indicates abnormal retinal correspondence. One cannot predict in which case normal and in which abnormal retinal correspondence will develop or be manifested.

THE CORNEAL LIGHT REFLEX AS AN AID TO MEASUREMENT OF SQUINT

I mentioned that squint may show either normal or abnormal retinal correspondence. The corneal light reflex is an infallible guide in measuring strabismus. The mere displacement of a corneal light reflex

is only of qualitative value and is therefore unsatisfactory for purposes of measurement. My reasons for this assertion were explained in a previous article.[3]

The accurate measurement of squint is a most important preliminary consideration in studies of fusion and retinal correspondence. It is axiomatic that a corneal light reflex is displaced in a squinting eye from its position of fixation. Any artificial means that could restore the position of the corneal light reflex to a central or fixational position also serves as a measure of the squint (fig. 3). The prism reflex test which I described has proved a method *par excellence* and can be used by any practitioner with but little practice. The refined Brewster stereoscope, the anglometer and the synoptophore are additional aids in the controlled examination of squint and of retinal correspondence. Unless targets are translucent and brightly illuminated to register satisfactory corneal light

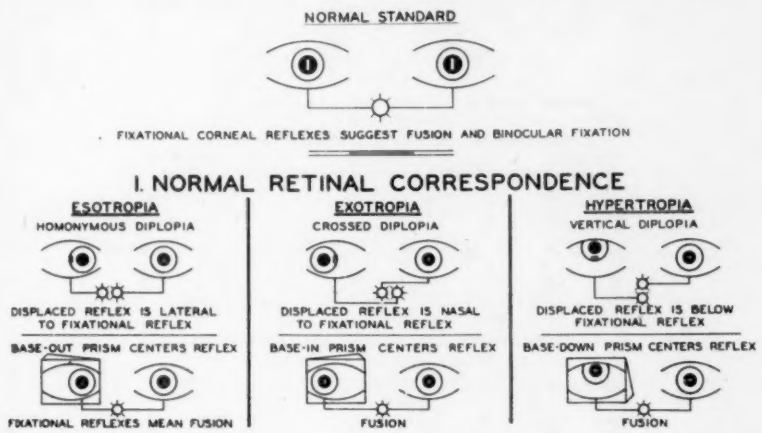

Fig. 3.—Binocular projection in relation to corneal light reflexes: normal retinal correspondence.

reflexes and, moreover, can be flexibly adjusted to correspond to the visual axes for the two eyes, no stereoscope is of value. The phorometric stereoscope provides a comprehensive method for studies not only at the equivalent range of infinity but at selected ranges of accommodation. Each of these three instruments employs the principle of independent light sources and an intervening septum or independent viewing arrangement. The anglometer, moreover, can be used without the septum in conjunction with the prism reflex test and the Maddox rod in selected cases.

3. Krimsky, E.: The Fixational Corneal Light Reflexes as an Aid in Binocular Investigation, Arch. Ophth. **30**:505-521 (Oct.) 1943.

RETINAL CORRESPONDENCE IN RELATION TO CORNEAL LIGHT REFLEXES

The position of the corneal light reflex is a dependable means of interpreting the direction or projection of the false image both in cases of paralytic diplopia and through artificial stimulation in cases of normal retinal correspondence. In abnormal retinal correspondence, the position of the corneal light reflex does not correspond to the position or direction of the false image produced by adequate artificial stimulation. Thus, the position of the corneal light reflex as an objective sign is of decisive value in differential diagnosis when compared with image responses (fig. 3). If one conceives of a central or a fixational position of the corneal light reflex as indicating projection forward, then any displacement of the corneal light reflex in relation to this fixational position should suggest false projection in a relative direction. When the

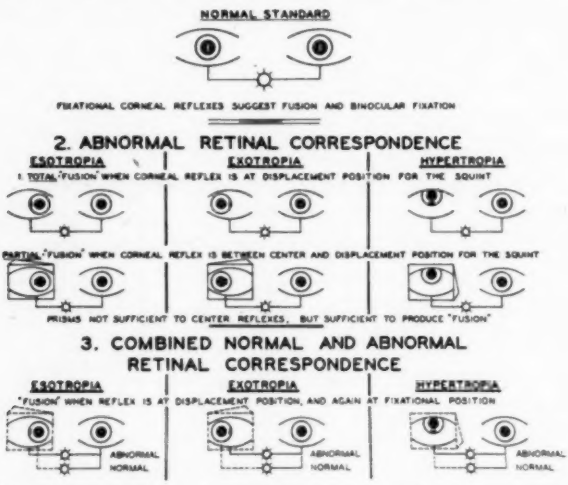
NORMAL STANDARD
FIXATIONAL CORNEAL REFLEXES SUGGEST FUSION AND BINOCULAR FIXATION
2. ABNORMAL RETINAL CORRESPONDENCE
ESOTROPIA
EXOTROPIA
HYPERTROPIA
1. TOTAL "FUSION" WHEN CORNEAL REFLEX IS AT DISPLACEMENT POSITION FOR THE SQUINT
2. PARTIAL "FUSION" WHEN CORNEAL REFLEX IS BETWEEN CENTER AND DISPLACEMENT POSITION FOR THE SQUINT
PRISMS NOT SUFFICIENT TO CENTER REFLEXES, BUT SUFFICIENT TO PRODUCE "FUSION"
3. COMBINED NORMAL AND ABNORMAL RETINAL CORRESPONDENCE
ESOTROPIA
EXOTROPIA
HYPERTROPIA
"FUSION" WHEN REFLEX IS AT DISPLACEMENT POSITION, AND AGAIN AT FIXATIONAL POSITION
ABNORMAL
NORMAL
ABNORMAL
NORMAL
ABNORMAL
NORMAL

Fig. 4.—Binocular projection in relation to corneal light reflexes: abnormal retinal correspondence.

reflex is displaced upward in relation to the fixational position, the false image is likewise displaced upward. When the reflex is displaced outward, the false image is outward, or homonymous. The reflex can be displaced outward only when the eye is deviated inwardly. The amount of displacement of the light reflex from the fixational position suggests the magnitude of separation of the true and false images. In normal retinal correspondence the projection of the false suppressed image would conform to such displacement of the corneal reflex. In abnormal retinal correspondence, on the other hand, even though the light reflex is displaced there is a tendency for approximation or superposition of the false and the true images at or near the primary, or binocular, angle (fig. 4).

NATURE OF FUSION IN ABNORMAL RETINAL CORRESPONDENCE

I stated previously that in abnormal retinal correspondence both the squinting and the fixing eye attempt under artificial conditions to project images registered in the brain to or near a common point, even though the corneal light reflex is displaced in one eye. This can be attained only when a paramacular area of the retina in one eye coordinates latently with the macula of the fellow, or fixing, eye, in other words, when there is a macular-paramacular association. In convergent squint, the corneal light reflex is displaced temporally but the registration on the retina is nasal, and the retinal correspondence would be macular-nasomacular. In divergent squint, the corneal light reflex is displaced medially and the image on the retina is received temporally, and the retinal correspondence would be macular-temporomacular. This paramacular area is sometimes erroneously termed the "false macula" because the image strikes an area other than the true macula (fig. 5).

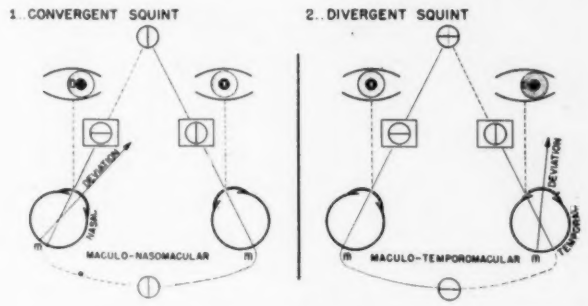

Fig. 5.—Association areas in squint with abnormal retinal correspondence.

Just as a squint may be alternating, so may this macular-paramacular association be alternating or transferable when either eye assumes the role of fixation (fig. 6).

In normal retinal correspondence, in contradistinction to abnormal retinal correspondence, the fusion obtained under artificial conditions is apt to be more critical because it is bimacular. It is usually a latent macular-macular relationship, which can be manifested only by artificial means, such as prism, stereoscope or synoptophore. A prism that can "correct" a deviation will often awaken bimacular registration, and even a latent fusion amplitude around this angle of deviation. With the stereoscope, too, such latent dynamic response can be induced when targets are brought into the range of the deviation. Such a dynamic response may confuse the examiner in establishing the angle of deviation; the von Graefe supplementary prism dissociation test prevents any possibility of fusion at the angle of the squint.

In abnormal retinal correspondence latent approximation or "fusion" of images is not bimacular in correspondence. Hence, no fusion amplitude can be induced through artificial means.

A latent area of relative suppression is usually associated with both normal and abnormal retinal correspondence accompanying squint. This suppression serves to absorb the spontaneous false image. It is relative in the sense that it occurs only when fixation is assumed by the fellow (fixing) eye and is not necessarily related to loss in monocular visual acuity. The area and the severity of this suppression vary in different instances. This area can be mapped out with use of targets that subtend minimal retinal angles by various binocular methods which permit the nondeviating eye to assume the role of fixation. Travers employed a Bjerrum screen for the nonfixing eye and a mirror device for the fixing eye and aimed to plot the macular position of the suppressing eye by suitable adjustment of the fixing eye.[4]

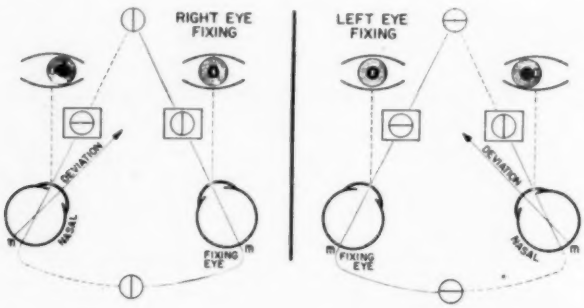

Fig. 6.—Abnormal retinal correspondence—an alternating phenomenon in a case of convergent squint.

RELATIVE SUPPRESSION OF SQUINT (FIG. 1)

A fused image of two separate targets as seen normally through a stereoscope has a more distinct component belonging to the dominant, or fixing, eye and a hazier one for the fellow, or so-called nonsighting, eye. The hazier component may prove blurred and indistinct in squint, owing to a larger zone of relative suppression, making it necessary to employ a larger target in order to register rudimentary fusion. When the zone of suppression is large and a smaller moving test object is brought gradually into the central binocular field, there will be sudden disappearance of image of this moving test object, only to have the image reappear as the target reaches the opposite side of the suppression area. This phenomenon is described as a "jump," or approximation, or crossing, of images without actual touching. The larger the target, the

4. Travers, T. a'B.: Suppression of Vision in Squint and Its Association with Retinal Correspondence and Amblyopia, Brit. J. Ophth. **22**:577-604, 1938.

less the chance of total fading. From the standpoint of the examiner, it is important to appreciate the physiologic basis of this phenomenon in evaluating painstaking measurements which sometimes seem to make no sense. It is also important to ascertain whether this relative suppression belongs to the macular or to the paramacular area. The relation of the zone of suppression to the position of the corneal light reflex seems to indicate whether it is part of normal or of abnormal retinal correspondence.

FALSE MACULA

False macula should be distinguished from so-called paramacula, which one usually associates with abnormal retinal correspondence. False macula is also termed eccentric fixation because the affected eye appears to be in a squinting position even though the other eye is covered (fig. 7). It is not uncommon in cases of unilateral squint, and because

Fig. 7.—Photographs of false macula, or eccentric fixation, in one eye.

the squinting eye suffers from disuse severe amblyopia is a common result. On alternate screening there appears to be no motion in either eye, such as one would expect in alternating squint. Vision is often reduced below 20/200, and when the affected eye is made to assume fixation by covering the good eye one often observes an unsteady nystagmoid movement, because monocular fixation is greatly impaired. Even in such cases of greatly reduced vision it is amazing to discover some form of suppressed or rudimentary correspondence of the two eyes.

Abnormal retinal correspondence, in contradistinction to so-called false macula or eccentric fixation, manifests good or passable vision in each eye, and monocular fixation is steady.

VARIATIONS IN ABNORMAL RETINAL CORRESPONDENCE

Abnormal retinal correspondence is not a clearcut entity so far as accuracy in measurement is concerned. It is clearcut only in the sense

that the "fusion" angle does not correspond to the angle of the deviation or to the fixational corneal light reflexes. Abnormal retinal correspondence may exist also in cases of vertical squint, in which artificially induced double images will appear on the same horizontal level even though the corneal light reflex and the eyeball are displaced vertically.

Abnormal retinal correspondence may, and often does, occur in association with normal retinal correspondence; and "fusion" or approximation of double images may be induced when the light stimulation corresponds to the displaced position of the corneal light reflex for the deviated eye and, again, when the light reflex is restored artificially (as by prism or stereoscope) to the central fixational positions for both eyes. The abnormal retinal component may be more prominent than the normal, and vice versa. As a rule, even when the abnormal element is intense, it is fairly easy also to awaken normal retinal correspondence by moving brightly illuminated stereoscopic targets in line with both visual axes or both fixational positions on the cornea. In thus awakening normal retinal correspondence, the abnormal component gradually becomes fainter, probably because the eyes prefer bimacular correspondence if that were only possible. During this course of stimulation the patient may encounter triplopia or even quadrilopia, which, fortunately, is transient.

When the induced images suggest superposition at the primary, or binocular, angle but with the corneal light reflex corresponding to the actual ocular deviation, abnormal retinal correspondence is said to be total, or harmonious.[5] Often, however, the image angle corresponds to a corneal reflex position somewhere between the fixational position and the maximally displaced position for the deviation. In such case, the abnormal component is said to be partial, or subharmonious (fig. 4).

HOW TO ELICIT RETINAL CORRESPONDENCE

If by retinal correspondence one means a bivisual response or projection based on ample light stimulation, then the positions of the corneal light reflexes should tell one whether such correspondence is normal or abnormal.

Any method or apparatus which can produce clearcut corneal light reflexes that can be flexibly displaced through such artificial means should prove ample for one's requirements. Such methods include use of (*a*) the prism reflex test, (*b*) the phorometric stereoscope, (*c*) the anglometer, (*d*) the synoptophore and (*e*) the maddox rod.

5. Worth, C.: Squint, edited by F. B. Chavasse, Philadelphia, P. Blakiston's Son & Co., 1939. Burri, C.: The Concept of Abnormal Retinal Correspondence, Arch. Ophth. **19**:409-424 (March) 1938.

THE ROTARY PRISM IN THE ESTIMATION OF RETINAL CORRESPONDENCE

In a previous article, I reported the use of the rotary hand prism or the phorometer for restoring the light reflexes to a fixational position for the objective study of squint. The additional application of the prism reflex test in the diagnosis and measurement of retinal correspondence is, I believe, a newer departure in prism testing (fig. 8).

The principle of this test is, first, artificial restoration of the corneal light reflex to a fixational position and, second, study of image responses after inducing diplopia with supplementary prism. Specifically, the test is carried out as follows:

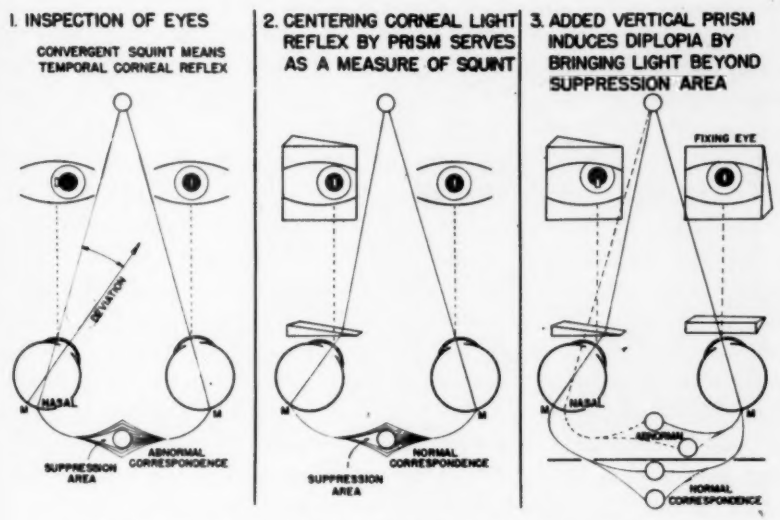

Fig. 8.—The phorometer in the study of retinal correspondence.

1. Observations on the eyes and the positions of the corneal light reflexes. The fixational corneal reflex may be central or off-center. Instead of measuring the angle gamma position, the examiner merely makes a mental note of the position of the corneal light reflex, first monocularly and then binocularly. With a little practice, the examiner can learn to tell at a glance the fixational positions without having to involve himself in needless computations. As long as binocular fixation is maintained, say, with convergence of the eyes or in overcoming the effects of a prism, the corneal light reflexes will remain in their respective monocular positions. The examiner holds the light directly in front of the patient's eyes and at a distance of 13 inches (36 cm.).

2. Measurement of the deviation by restoring fixational corneal light reflexes. The rotary hand prism is held before the deviating eye and

manipulated so as to produce movement of the corneal light reflex. If the corneal light reflex is temporal, as in convergent squint, a base-out prism before the deviating eye will restore the light reflex to a central, or fixational, position. If one thinks in terms of the apex of the prism and compares it to the point of an arrow it will be easy to figure out how to rotate the prism in order to centralize the corneal light reflex, because the point of the arrow will be in a direct line from the displaced position of the light reflex to the fixational position. If the examiner makes the mistake of rotating the prism in the wrong direction, the light reflex will move away, rather than toward, the center. The prism is kept free from dust, and the light reflex is observed through the prism. The amount of prism required to bring the light to the fixational position is the measure of the amount of squint. When the examiner believes that he has centered these light reflexes, he supplements with alternate screening to insure that motion has been stopped. If the rotary prism is placed before the fixing eye instead of the deviating eye, the light reflex will move in the direction of the apex of the prism. Such movement will also bring the deviating eye out, and here, too, the corneal light reflex will be gradually seen to become centered.

3. Image Response. Is there a latent tendency to normal or abnormal retinal correspondence? Usually the patient continues to see single as the corneal light reflex is gradually centered because of latent suppression in one eye. By inducing diplopia after reflexes are centered, one can determine whether retinal correspondence is normal or abnormal. In normal retinal correspondence images approach or cross at or near the bimacular angle corresponding to the fixational corneal light reflexes. In cases of horizontal squint my associates and I induce vertical diplopia after the corneal light reflexes are restored to fixational positions by rotary prism. A supplementary prism of 10 Δ base down over the eye not covered by the rotary prism (fig. 9) will be sufficient to overcome suppression and induce diplopia. If necessary, a stronger prism may be required. The corneal light reflex will be in a central, or fixational, position in one eye and displaced vertically in the other eye. Such artificial vertical displacement of the corneal light reflex in one eye by prism likewise registers true vertical diplopia in normal retinal correspondence. In abnormal retinal correspondence double images appear oblique because association is macular-paramacular. In other words, the subjective angle does not correspond to the objective, or deviation, angle. By rotating prism to bring images into true vertical alinement, we now obtain a reading for the subjective angle, but such operation of the prism has now altered the position of the corneal light reflex away from the objective angle. An appreciable disparity in prism readings for subjective and objective angles points to abnormal retinal correspondence. Prism readings for the image, or subjective, angle (true vertical

diplopia) will always be found nearer the 0 position than the reading for the deviation, whether it be an esotropia or an exotropia.

In vertical squint we study image responses by inducing diplopia with base-in or base-out prisms. When such induced double images are found to be horizontal in the presence of vertical deviation and vertical displacement of the corneal light reflex, the condition points to abnormal retinal correspondence. Furthermore, the eyes will be seen to move up and down to meet images that appear on the same level. In normal retinal correspondence such induced double images will be found on different levels, and "correction" of the vertical squint by vertical prism sufficient to bring the corneal light reflex to the fixational level will likewise level the images and overcome up and down movements of the eyes to meet the images.

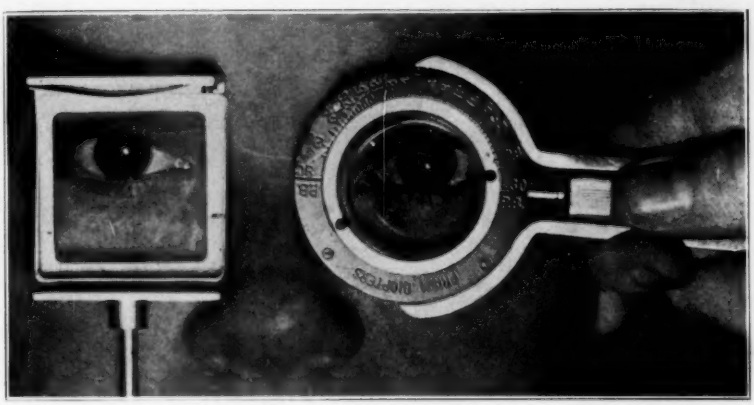

Fig. 9.—Krimsky prism holder and rotary prism.

THE MADDOX ROD AND RETINAL CORRESPONDENCE

The Maddox rod may be used as a substitute for the von Graefe dissociation test. After the corneal light reflex is restored by prism to a fixational, or bimacular, position in the deviating eye, the Maddox rod is then placed over either eye; in cases of horizontal squint the rod is placed horizontally to produce a vertical streak and in cases of vertical squint it is placed vertically to produce a horizontal streak (fig. 10).

Normally, the image of the streak should meet the spot of light at the bimacular angle when both corneal light reflexes have been made fixational. In orthophoria superposition is automatic. In heterophoria or in normal retinal correspondence such approximation can be obtained only when the corneal light reflex in the deviating eye is brought into a fixational position by means of rotating prism. Inspection of the

corneal light reflex behind the Maddox rod may not be possible. However, if the examiner instructs the patient to fix his attention on the streak rather than on the spot of light the eye behind the Maddox rod will be brought into a fixational position and displacement of the reflex in the uncovered, or nonfixing, eye can therefore be observed and studied. Instead of alternate screening to determine when motion of the eyes has finally stopped with sufficient prism, the examiner may simply ask the patient to fix alternately the streak and the spot of light and then note when motion has been arrested.

In normal retinal correspondence superposition of light and streak occurs when the corneal light reflexes have been brought to the fixational corneal positions and when alternate fixation produces no movement of the eyes. In abnormal retinal correspondence approximation of light and streak will occur in spite of displacement of the corneal light reflex

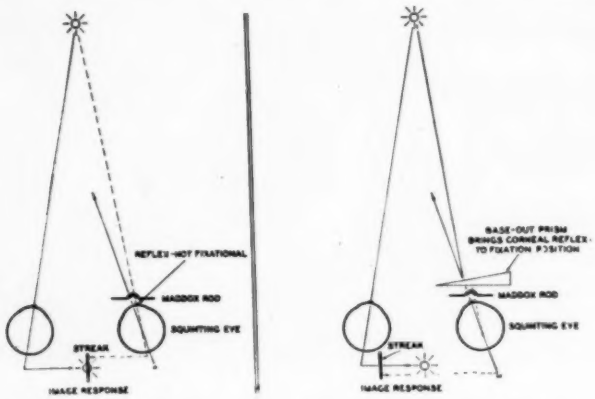

Fig. 10.—The Maddox rod in the study of retinal correspondence. The rod is held at a 13 inch distance.

in one eye, and alternate fixation of streak and light will still yield appreciable movement of the eyes in order that the patient may see images which appear very close together. In other words, the image angle does not correspond to the angle of deviation, or objective angle.

In cases of vertical squint, the Maddox rod is placed vertically before either eye to produce a horizontal image of the streak. With both eyes open, the streak will appear higher or lower than the spot of light in normal retinal correspondence; and when the vertical deviation, as well as the vertical displacement of the corneal light reflex, is "corrected" by vertical prism, both the spot of light and the streak will appear leveled and there will be no up and down movement of the eyes on alternate fixation of respective images. In cases of abnormal retinal correspondence, whether total or partial, when images are found or made level

by prism that does not fully correct, the corneal light reflex will still be vertically displaced and there will be appreciable up and down movement of eyes on alternate fixation of light and streak.

Incidentally, I rely on the movements of the lids, as well as on movements of the eyes. When the eyes move exactly up and down, the lids will respond in the same way. The slightest oblique movements of the eyes will show themselves in analogous excursions of the lids.

THE ANGLOMETER IN THE DIAGNOSIS OF RETINAL CORRESPONDENCE [6]

The anglometer is an arclike arrangement pivoted on the sides rather than in the center, as in the perimeter. The entire arc, therefore, can be made to move up and down to correspond to the upward and downward movements of the eyes. Its movable lighted targets are flexibly moved along the arc into any desired testing position to the right or the left for any selected level of gaze. Each target carrier,

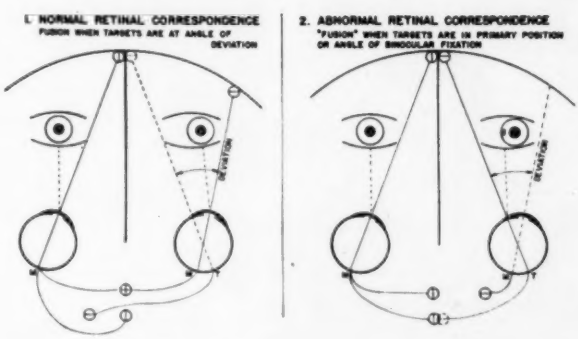
1. NORMAL RETINAL CORRESPONDENCE
FUSION WHEN TARGETS ARE AT ANGLE OF DEVIATION
2. ABNORMAL RETINAL CORRESPONDENCE
"FUSION" WHEN TARGETS ARE IN PRIMARY POSITION OR ANGLE OF BINOCULAR FIXATION

Fig. 11.—The anglometer in the measurement of divergent squint and retinal correspondence.

moreover, permits independent raising or lowering of the test light to correspond to the vertical, as well as to the horizontal, deviation. Not only are such deviations noted readily in relation to a fixed position for either eye, but the examiner is also in a position to know exactly how many degrees upward or downward such measurements are made. Alinement of the targets to the deviation of one eye can be determined at a glance when the corneal light reflexes have been moved into their respective fixational positions and ocular movements have been arrested by alternate flashing or fixation. In normal retinal correspondence the image angle represented by fusion or approximation of images corresponds to the deviation reading, as determined in the preceding section. In abnormal retinal correspondence superposition or crossing

6. Krimsky, E.: The Cardinal Anglometer, Arch. Ophth. **26**:670-674 (Oct.) 1941.

of images will occur at a different setting, which will require further movement of targets in order to bring such images together. At this new, or subjective, angle the light reflex will no longer be in the fixational position on one eye, and the eyes will be seen to move appreciably from one image to the other, even though they appear to be touching or very close together. In such cases, the subjective (or image) angle and the objective angle do not correspond, and one notes the two different readings (fig. 11).

In measuring deviation, the examiner instructs the subject to maintain fixation on one of the targets. He then disregards that eye as well as

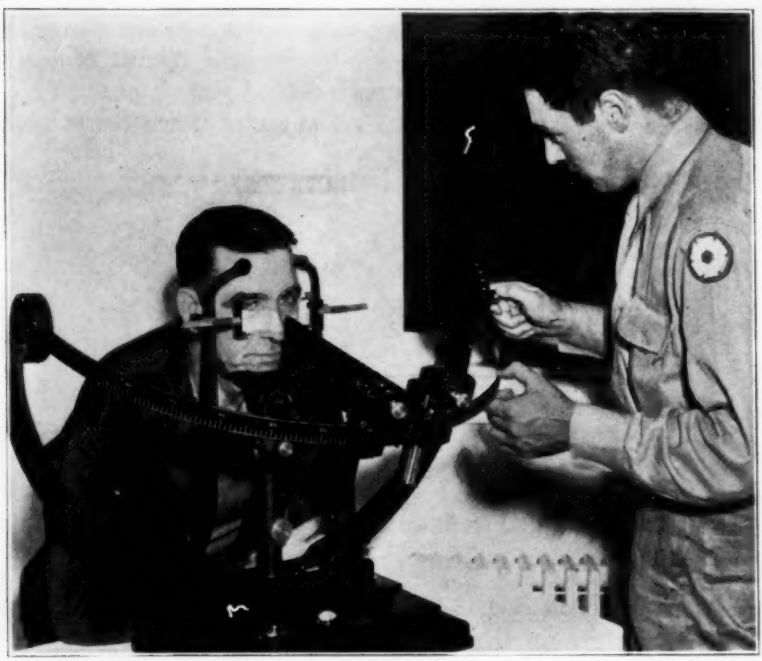

Fig. 12.—The cardinal anglometer.

that target and merely operates the target to the deviating eye so as to restore the light reflex to a fixational position. In moving such a target, he keeps his eye at all times behind the light, so as to obtain the true effect. He first corrects the horizontal component of the deviation by moving the target carrier along the arc. Often, with a little experience he will arrive at the correct reading and will note the angular deviation on this calibrated arc, which he will record in relation to the position of the fixing eye along the arc. There may be an additional vertical disparity, and the target holder enables the test light to be raised or lowered and vertical readings also to be noted in degrees. In addition

to restoration of fixational corneal light reflexes, he looks for cessation of ocular movement on alternate fixation or flashing. He also notes image responses. If the two lights appear fused or near together at this deviation reading, normal retinal correspondence is indicated. If another setting of targets is required to produce superposition of images, the examiner now notes the reading for the superposition or image angle, as well as the reading for deviation angle, in terms of abnormal retinal correspondence.

Examination with the anglometer is simple and direct and conforms to accepted principles of binocular study. Because there is no collimating lens, the findings conform more closely to what one would expect with the cover test. In other words, it aims to serve as a quantitative cover test for a fixed distance of 13 inches (33 cm.), but with

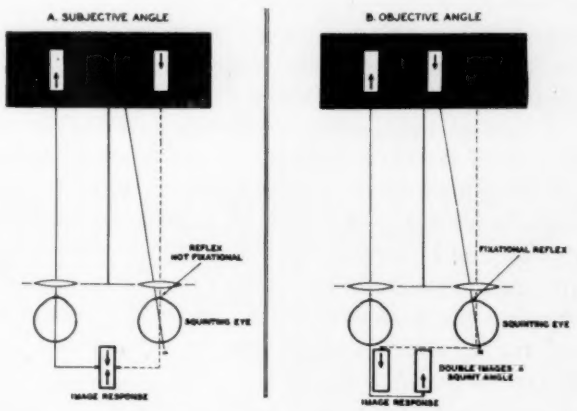

Fig. 13.—The stereoscope and use of displaced targets in the study of retinal correspondence.

the added advantage that the examiner can readily test the eyes for any measured and selected position of gaze without worrying about instinctive movements of the head. Control of the fixational corneal light reflex is most satisfactory and is unhampered by prisms or lenses. Because study of cardinal or extraprimary positions is limited to a distance of about 13 inches, the test fulfills the requirements for investigation of the motor fields (fig. 12). The anglometer, by virtue of its arclike arrangement, yields readings in arc degrees and can be adjusted readily to selected positions of the eyes in a measured direction of gaze and with fixed control of the head; the phorometer, or rotary prism, is essentially a phoria instrument which provides readings in prism diopters for the primary position of gaze and is limited to the lesser degrees of squint.[6]

THE PHOROMETRIC STEREOSCOPE AND RETINAL CORRESPONDENCE

The Brewster type of stereoscope has carried with it an unfortunate prejudice, namely, that it is thought to be inadequate in the diagnosis of ocular deviation and retinal correspondence by those trained in the Wheatstone school. It is true that the Wheatstone type of stereoscope or synoptophore has been improved for such studies. However, the simpler type of Wheatstone instrument or Worth amblyoscope, without a calibrating system and effective illumination, is not much more satisfactory than the simpler type of Brewster stereoscope. A distinction is made between the major and the minor amblyoscope. The Brewster instrument is still excluded as a minor, even though it has been dressed up by commercial outfits with elaborate flashing units, modernistic designs and other features to detract one from the idea that it could still be a simple instrument bidding for popularity.

To transform a Brewster instrument into a clinical instrument, it was necessary to introduce the following changes: (1) brightly illuminated, transparent targets, which yield clear corneal light reflexes; (2) ease in inspection of corneal light reflexes through a reflecting mirror; (3) reduction in size of targets to permit greater range of amplitude; (4) calibrating arrangement, which automatically adjusts the targets to the primary positions for selective ranges of accommodation; (5) elimination of the antiquated caboose-like viewing head to permit increased flexibility in accommodation and to keep the eyes exposed; (6) provisions for supplementary square prisms, and (7) increase in the area of peripheral retinal stimulation and in the light intensity by merely bringing the targets closer to the eyes.

Through such improvements,[7] it has been possible to adapt this type of instrument to the large majority of patients with convergent squint up to at least 70 Δ both for the 0 and for the 3 D. range of accommodation. For larger readings one must think surgically, and if exactness in measurements is desired an arclike apparatus appears more satisfactory.

In measuring deviation with the stereoscope, the patient maintains fixation of one eye on the corresponding target and the position of the corneal light reflex is observed in the nonfixing, or deviating, eye. The targets can be approximated ("converged") or separated ("diverged") mechanically, and corresponding displacement of the corneal light reflex is observed through the mirror. As soon as the reflexes appear in the fixational positions, alternate flashing or fixation of targets is done to observe whether movement of the eyes has been arrested. Image response is also studied; that is, it is noted whether the two images meet closely at this angle of deviation or appear widely separated. Super-

7. Krimsky, E.: Modification of the Brewster Stereoscope for Clinical Requirements, Arch. Ophth. **26**:808-815 (Nov.) 1941.

position or approximation of images at this objective angle speaks for normal retinal correspondence. Wide separation of images requiring further displacement of targets to produce such superposition brings them out of alinement with the angle of deviation and hence speaks for abnormal retinal correspondence.

CONCLUSIONS

1. Abnormal retinal correspondence is a compensatory rudimentary macular-paramacular association between a fixing and a squinting eye.

2. Only under artificial conditions can such a latent partnership be demonstrated, because a relative suppression exists in the nonfixing eye.

3. Retinal correspondence, whether normal or abnormal, must have a psychic association; otherwise there can be no functional retinal correspondence.

4. Normal retinal correspondence is not necessarily, as its name implies, a normal condition. It merely indicates fusion or approximation of images which strike corresponding macular areas of the retina. Such correspondence occurs normally and spontaneously in normal binocular fixation and artificially in cases of squint.

5. An accurate measurement of ocular deviation is a necessary preliminary to the study of retinal correspondence. Artificial restoration of the corneal light reflex to a central, or fixational, position is, in my opinion, the most reliable method of measuring accurately ocular deviation.

6. The displaced corneal light reflex is dependable only so far as one can judge whether deviation is present. Estimation of deviation based on mere inspection of the position of the displaced reflex appears crude and inaccurate.

7. The prism reflex test is a gratifying method for centralizing the corneal light reflex. The amount of prism required to center the light reflex is, likewise, a measure of the amount of squint. When combined with the screen or cover test, it is confirmatory.

8. To interpret squint solely on the basis of deviation implies disregard of functional and psychologic responses. Every patient who has squint with vision in each eye has some form of retinal correspondence.

9. The position of the displaced corneal light reflex in relation to the position of the fixational reflex tells one at a glance the projection of the false image in paralytic squint and in normal retinal correspondence.

10. One cannot tell from the mere appearance of a squint whether there is normal or abnormal retinal correspondence or both. Some form

of retinal correspondence or rudimentary fusion exists in practically every case of squint without a pathologic condition.

11. One should think of fusion response in terms of gradation, much as one thinks of visual acuity.

12. The fusion which occurs in normal binocular fixation is sufficiently secure to enable amplitude or flexibility in both the convergence and the divergence range. Fusion in manifest squint is suppressed but can usually be awakened in some degree by adequate artificial stimulation.

13. In normal retinal correspondence there is often produced a fusion amplitude in the region of the deviation; in abnormal retinal correspondence there can be no fusion amplitude because correspondence is not bimacular.

14. The "fusion" that can be awakened in squint by brief artificial stimulation or "exercise" reverts to its status of inertness or relative suppression unless the position of the eyes can also be rectified.

15. Abnormal retinal correspondence is subject to variations. It may be total or partial, vertical or horizontal, or it may be combined with normal retinal correspondence. Furthermore, responses vary with different tests.

16. The diagnosis of abnormal retinal correspondence does not belong to the province of orthoptics. It is as intimately associated with clinical ophthalmology as is refraction or perimetry, and its interpretation is part of medical and surgical judgment. The diagnosis of retinal correspondence is more important than its actual measurement. Exactness in measurement is desirable, but flexibility in judgment is even more important.

17. The subject of normal and abnormal retinal correspondence offers a field for intriguing clinical research. Measurement of deviation alone is merely an introduction to the study of squint and therefore tells but half the story.

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TRANSPLANTATION OF THE VITREOUS

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THIS experimental study was embarked on to determine the feasibility and limitations of transplanting the vitreous. The vitreous has proved to be one of the most difficult parts of the eye to deal with, and has been approached by a small number of workers in different ways, with scant success. The experimental studies on this structure have been remarkably few considering its importance and volume, probably because most ophthalmologists have a great fear of the vitreous and want to stay away from it. It does not take an ophthalmic surgeon long to learn that it is desirable never to see the vitreous, as its loss, injury or infection is poorly tolerated. The diseases of and degenerative changes in the vitreous are subtle and are not well understood; worst of all, they do not respond to the treatment heretofore available.

Withdrawal of the vitreous as a therapeutic measure was performed by Ford [1] and zur Nedden.[2] The latter found that he could remove up to 0.6 cc. without apparent damage, and even with benefit in some cases. Deutschmann [3] performed the first recorded transplantation of vitreous from calf and rabbit eyes into the human eye. Komoto,[4] Löwenstein and Samuels [5] and Elschnig [6] removed vitreous and replaced it with saline solution. A number of men, among them Elschnig, have

The work was done under a grant from the William L. Hernstadt Fund in the Ophthalmic Service at Montefiore Hospital for Chronic Diseases.

1. Ford, V.: Proposed Surgical Treatment of the Opaque Vitreous, Lancet **1**: 462, 1890.

2. zur Nedden, M.: Ueber den Heilwert der Punktion des Glaskörpers, Arch. f. Ophth. **101**:145, 1919-1920; Das Instrumentarium zur Glaskörperabsaugung, Klin. Monatsbl. f. Augenh. **66**:474, 1921; Ueber Glaskörperabsaugungen, ibid. **67**:305, 1921; Lanzenkanulen zur Glaskörperabsaugung, ibid. **69**:514, 1922.

3. Deutschmann, R.: Zur operativen Behandlung der Netzhautablösung, Klin. Monatsbl. f. Augenh. **4**:364, 1906.

4. Komoto: Ueber Glaskörperwaschung bei unheilbarer Glaskörperblutung, abstracted, Klin. Monatsbl. f. Augenh. **50**:265, 1912.

5. Löwenstein, A., and Samuels, B.: Ueber Glaskörperersatz, Arch. f. Ophth. **80**:500, 1911.

6. Elschnig, A.: Ueber Glaskörperersatz, Arch. f. Ophth. **80**:514, 1911.

injected air into the vitreous. Cutler [7] recently reported the transplantation of human vitreous from one patient to another in 3 cases, with a good visual result in 2 cases, though all the patients appeared blind before operation as a result of hemorrhage or infection of the vitreous. We were not aware of Cutler's work until the present study was almost ready for publication, and were extremely interested in his report.

PRESENT INVESTIGATION

Our studies were performed on the eyes of young albino rabbits. A number of difficulties were encountered and a number of eyes lost by following technics described by previous workers. Intraocular hemorrhage at the time of operation occurred in almost every case. A few days after the operation opacities of the vitreous and lens were seen in many of the eyes.

The rabbit eye proved more difficult to manipulate than the human eye, owing to the large lens in the small eye with a small vitreous space. The anteroposterior diameter of the young rabbit eye is 14.5 mm., as compared with 25 mm. for the human eye. In the rabbit eye there is only 3.5 mm. of vitreous space between the posterior surface of the lens and the retina, as compared with 16 to 17 mm. in the human eye. There is little leeway, therefore, in entering the vitreous space in the rabbit eye, and great care must be exercised to avoid damage to the lens and the retina.

We thought that we should eliminate the danger of retinal detachment by sealing the retinal holes with diathermy; so preliminary surface diathermy was applied. However, we soon found that we could dispense with this procedure, since holes can be found in the rabbit's retina without a detachment developing. In 36 rabbits, on some of which we made repeated punctures, a permanent retinal detachment developed in only one eye. However, we should not dare to puncture a human retina without using diathermy to seal the holes, as experience with perforation of foreign bodies has demonstrated a greater tendency to detachment.

In all these procedures surgical asepsis must be maintained. Local and general anesthesia were used, but we soon found general anesthesia induced with pentobarbital sodium most convenient to employ.

As stated previously, simple withdrawal of vitreous had been done before, but we thought it of interest to repeat this in the rabbit. If no more than 0.3 cc. of vitreous was withdrawn, there was only a transient conjunctival flush, and the globe was not altered in size or shape. Hypotony followed the withdrawal of vitreous but was of only

7. Cutler, N. L.: Transplantation of Human Vitreous, Arch. Ophth. **35:** 615 (June) 1946.

short duration. The media stayed clear, and no retinal damage was noted.

After the withdrawal of 0.4 cc. or more of vitreous, the signs of disturbance became progressively severer, and the final results were disastrous. Hemorrhages appeared on the iris and retina and occasionally in the vitreous. Withdrawal of 0.9 cc. of vitreous caused the eye to collapse like an empty tobacco pouch, so that the needle point almost invariably damaged the retina, and often the lens. The conjunctiva became engorged and the vessels of the iris prominent, and blood appeared in the anterior chamber. The eyeball gradually regained its shape, but the eye did poorly and usually degenerated.

Histologic examination of eyes from which 0.9 cc. of vitreous was withdrawn and the eye immediately enucleated disclosed the extensive damage that occurs on withdrawal of the vitreous in this manner. The intraocular vessels were extremely dilated, and there were extensive hemorrhages in the anterior chamber, and into the iris, the ciliary body and the vitreous. The choroid was detached as a result of hemorrhage and an accumulation of serous fluid; the retina was detached owing to hemorrhage.

This histologic study revealed the reasons for some of the difficulties encountered in transplantation of vitreous. The sudden withdrawal of a large amount of vitreous causes collapse of the globe and a sharp drop in intraocular tension, with loss of support for the blood vessels. This produces a high degree of hyperemia and hemorrhages, with resulting rapid and profound damage to the eye. Of course, the hemorrhages can be absorbed, but our experience with the eyes which were permitted to recover was not encouraging.

The next studies consisted in removing vitreous in varying amounts and then replacing it with vitreous from the other eye, using a technic of withdrawal and subsequent injection similar to that described by Elschnig and others and recently by Cutler. However, we found this method rather unsatisfactory. The damage immediately following withdrawal of large amounts of vitreous, causing the globe to collapse, was so great that replacement of the vitreous, even within a minute, was not enough to correct it. Large hemorrhages in the iris, retina and vitreous followed, so that most of the eyes were lost. However, there was enough evidence to encourage us in the belief that the transplantation of vitreous was feasible.

It became clear that it was important to avoid the collapse of the globe and the high degree of hypotony that followed withdrawal of large amounts of vitreous. A double needle and syringe was devised to permit simultaneous injection and withdrawal of vitreous. The mechanism consists of two needles soldered together, with their openings facing in opposite directions, and an arrest to stop the penetration

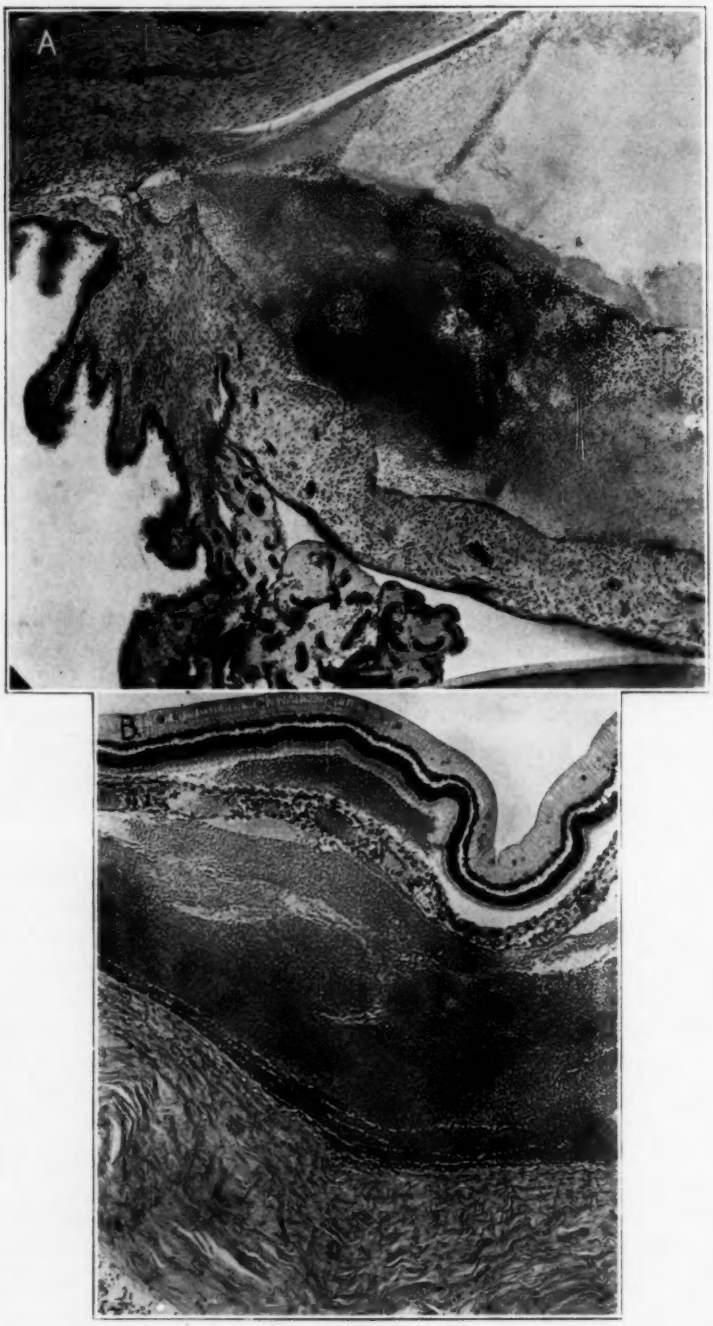

Fig. 1.—Rabbit eye from which 0.9 cc. of vitreous was withdrawn. (*A*) the the anterior chamber contains a large hemorrhage, and the iris and ciliary processes are extremely hyperemic (magnification, 30); (*B*) subretinal and subchoroidal hemorrhages have detached the retina and choroid (magnification, 50).

of the needle at the level desired. 'One syringe is filled with vitreous to be injected; the other is empty. The double needle is inserted through the sclera up to the arrest and one operator injects vitreous, while the other withdraws vitreous from the eye.'

'The vitreous is a colloidal gel, which liquefies only too readily. The larger the bore of the needle through which it passes, the less it is injured. However, the larger the needle, the more damage is done to the eye. After considerable experimentation, it was found that use of the 18 gage needle was the best compromise for the rabbit eye.'

'With this method of simultaneous withdrawal and injection, we immediately noted a dramatic improvement in our results and found that transplantation of vitreous in the rabbit eye was easy and well tolerated, without untoward developments, in almost every case.' There was no longer seen the hyperemia and hemorrhage in the iris, or even much conjunctival reaction. An occasional small hemorrhage was noted in the retina or vitreous, as well as occasional opacities in the vitreous; in only 1 eye was there retinal detachment, and in that eye scleral

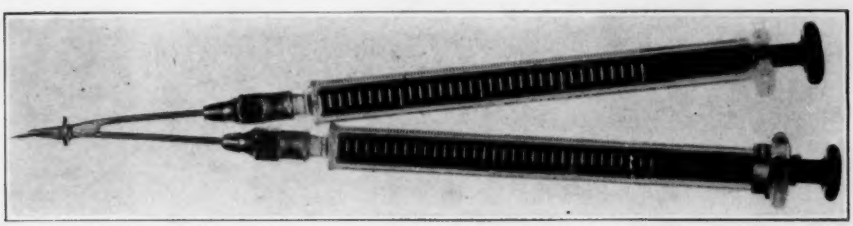

Fig. 2.—Double needle for transplantation of the vitreous to permit simultaneous injection and withdrawal of vitreous.

diathermy had not been used. The intraocular tension was maintained fairly well throughout the procedure, a factor which is important in maintaining the form and function of the eye.

'In some eyes saline solution was substituted for vitreous, but these eyes did well only if a small amount was used.' With the substitution of larger amounts of saline solution, for vitreous, the eye did poorly. 'The best substitute for vitreous was found to be vitreous from another eye, and amounts up to 1 cc. were successfully transplanted.'

'It made no appreciable difference whether we transplanted vitreous from the other eye of the same animal or from the eye of another rabbit. In 3 cases we transplanted vitreous from a recently enucleated human eye to the rabbit eye with complete success. The vitreous of a kitten's eye was transplanted to the rabbit eye without any disturbing reaction. The tolerance to transplantation of vitreous from other species was greater than was expected.'

Histologic studies of a number of eyes were made at varying intervals after transplantation of vitreous. The puncture wounds of the sclera healed well. The edges of the retina curled up and the retinal hole remained, but there was no tendency to detachment. No significant damage was observed in the rest of the eye. The absence of degenerative changes after the extensive replacement of vitreous seemed significant and indicated the tolerance of the rabbit eye to the procedure.

Blood was injected into the vitreous and withdrawn at various intervals. The blood-vitreous mixture was withdrawn readily in a vitreous transplantation four hours after injection. When the blood stayed in the vitreous for several days, it coagulated in firm masses,

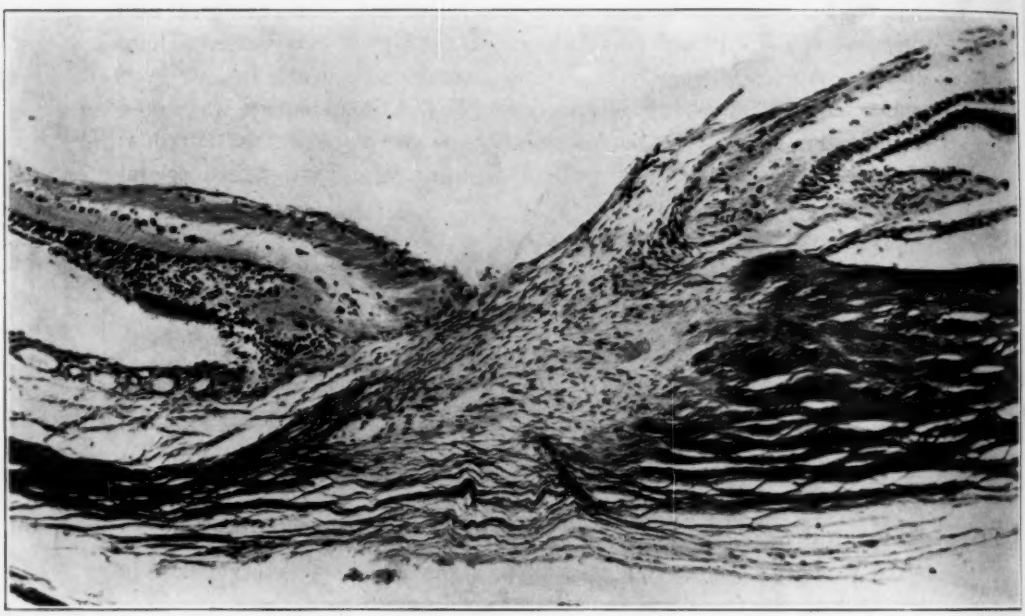

Fig. 3.—Eye of an albino rabbit which had been punctured with the double needle for transplantation of the vitreous, showing the retinal and choroidal hole and the healing of the scleral wound with connective tissue (magnification, 80).

which could not be withdrawn. In such eyes connective tissue bands formed in the vitreous, and later complicated cataracts developed. Apparently, blood must be withdrawn from the vitreous while still fluid. In the rabbit, a heavy coagulum forms rapidly and makes withdrawal difficult. Our impression is that in the human eye this coagulum does not develop so readily and may make transplantation of vitreous following hemorrhage more successful, as indicated by Cutler's [7] work.

With careful asepsis, we were able to perform a large series of transplantations of vitreous, with infection in only a few cases. The

vitreous is intolerant to infection; so unusual care is necessary to avoid contamination. Preservatives, such as penicillin and chlorobutanol, were tried; but they were not found to be superior to observance of a careful technic. The addition of chemical substances also raised concern over a possible alteration of the vitreous.

The vitreous was preserved by refrigeration at O C. for varying periods up to one week. During that period it lost some of its clarity and became clouded, though it was found that it could be injected without apparent harm after such preservation.

There are many uses to which transplantation of the vitreous may be put; the following are noted only as suggestions, and further study will be required before they are applied to the human eye: (1) to replace bloody vitreous; (2) to replace degenerated vitreous, as in cases of high myopia or in any case with opacities of the vitreous which obscure vision; (3) to restore vitreous lost in operation or through injury; (4) to restore the volume of the eye and its normal tension after operations for retinal detachment; (5) to relieve hypotony from any cause, and (6) to replace infected vitreous, possibly with the addition of antiseptics and antibiotics.

SUMMARY

Transplantation of vitreous has been successfully performed in rabbit eyes. Vitreous from the other eye of the same rabbit or from the eyes of other rabbits was used, as well as vitreous from the human eye and from the kitten eye.

A method of simultaneous injection and withdrawal of vitreous with a specially devised double syringe and needle arrangement was the only method found to give regularly successful results.

Further studies are needed before the method is applicable to the therapeutic uses suggested in the human eye.

Miss Hilda Bergen assisted in these studies and made the microscopic sections.

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USE OF THE TELESCOPIC AMBLYOSCOPE IN VISUAL TRAINING FOR DEFECTIVE VISION

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THIS paper is a preliminary report on the prognosis and treatment of a number of patients with ametropia, with vision ranging from 20/40 to 20/70 (amblyopic types) and higher refractive errors. All these patients had no definite pathologic condition of the eyes to account for the low vision, nor could the visual defect be corrected with glasses.

With the routine treatment as practiced in the clinic, if the patient was a young child, the mother was advised either to use atrophine or to keep a continuous patch on the good eye, depending on the degree of amblyopia. The purpose of this measure was to encourage the eye with the poorer vision to function properly. If with this treatment several months elapsed with no visual improvement, the case was placed in the category of congenital amblyopia, with its accompanying poor prognosis.

Occasionally the patient was unaware of his poor vision because there were no subjective symptoms. Some persons knew they had a visual defect but neglected the condition. With other patients the ophthalmologist may have felt that since the visual acuity could not be improved the patient had better not become acquainted with his shortcoming lest he become apprehensive of some further ocular disaster. Since the situation could not be improved, the physician might reason that "what the patient doesn't know, won't hurt him" and that matters had best be left alone.

Many patients became truly aware of the condition only after a thorough physical examination in a military, health or life insurance survey, which included a thorough examination of the eyes.

In this investigation it was determined that the first series of patients for study and treatment should consist of a sufficient number of adults and children to permit determination of the possible merit of treatment with the telescopic amblyoscope. The series included a few patients with amblyopia previously treated unsuccessfully by atropinizing and patching the good eye. Some were amblyopic patients referred by other

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ophthalmologists. The remainder were new patients whose vision could not be brought up to normal in spite of prolonged treatment with patching or atropinization of the normal eye, as well as reading exercises.

PRELIMINARY EXAMINATION

Vision of each of the first 19 patients to be examined was taken with three types of visual acuity charts. These patients, in addition, had their visual fields taken on the perimeter and the tangent screen. Each patient under 40 years of age had an ophthalmoscopic and a retinoscopic examination with use of a mydriatic. With patients over 40 the mydriatic was not used. Each patient was given the best possible correction. The glasses were worn for several months before visual training was suggested. Any muscle imbalance present was noted and treatment instituted. Other instruments available in the orthoptic clinic were made use of in the study and treatment of the patient if such a procedure was thought beneficial.

Of the 38 fields mapped for the first 19 patients examined, 8 presented mild contraction on the perimeter, and none showed any disturbance of the Mariotte blindspot. A few showed suppression scotomas.

By and large, the perimetric and tangent screen tests were not found to be of particular value, whether in diagnosis, prognosis or treatment. This was in accordance with findings in a previous study.[1] These tests were, therefore, omitted in the study of the succeeding cases. Central scotoma, if present, could usually be recognized by the eccentric fixation without perimetric studies as pointed out by Eggers.[2]

After the preliminary study only 10 of these 19 patients began the treatment schedule; they are included in the series of 47 patients reported here.

SUPPRESSION AS RELATED TO AMBLYOPIA

Suppression is a common obstacle in both good and poor vision. It is particularly frequent with squint but may also be present in persons without refractive error or squint. A review of the records of 39 patients treated at the squint clinic in the recent past showed that all had suppression. The suppressing eye had the higher error of refraction. To get an idea of how often persons without squint have suppression, 50 consecutive nonsquinting patients of various ages were examined. They had no particular ocular symptoms except those of eyestrain, or they came for an ophthalmic examination only because it was their habit to have their eyes examined every two years to make sure that their vision was normal. All those who were ametropic had their vision corrected to 20/20 with glasses before any tests for suppression were made. Some of the patients did not require glasses. In checking on the frequency of suppression in this group it was found that only 19 patients (38 per cent) had no suppression whatever. The other 62 per cent had suppression of various types. Fifteen patients (30 per cent) of the

1. Feldman, J. B., and Taylor, A. F.: Obstacle to Squint Training—Amblyopia, Arch. Ophth. **27**:851 (May) 1942.

2. Eggers, H.: Estimation of Uncorrected Visual Acuity in Malingerers, Arch. Ophth. **33**:23 (Jan.) 1945.

total series had alternating suppression of varying degrees, from occasional to mild to moderately strong or severe, even though the original refractive error was in some cases equal to that of the patients in the same group who did not suppress. Ten patients (20 per cent) also had suppression of varying intensity of the right eye only, while 6 patients (12 per cent) had suppression of the left eye of varying degree. When there was absolute suppression of an eye, that eye had the higher refractive error. Even at that, vision in all eyes, whether suppressing or not, was corrected to 20/20. On examination a few patients were found to have suppression but did not require glasses.[3]

Even from this small series it must be quite evident that suppression is a condition common to human beings regardless of their visual acuity and does not play an important role in the causation of amblyopia. It was mentioned previously [1] that Vogt and Grandi asserted that ametropia is not the absolute forerunner of amblyopia, as was previously assumed.

Even after the study of this small group, one is inclined to feel that suppression, like ametropia, plays a small part in either the cause or the effect of amblyopia. However, when suppression is severe, a particular effort should be made to overcome it lest improvement of the amblyopia be delayed. Prognosis in a case of amblyopia with severe or absolute suppression should not be given until the suppression is somewhat overcome.

INSTRUMENT USED FOR TRAINING VISUAL ACUITY

The device can be easily and cheaply constructed. It is built like a telescope, with an eyepiece and an objective, and is uniocular. It is more accurate than the Wollensak Allscope,[1] since it is calibrated. It can be more easily handled by the technician. It can be used like the Wollensak Allscope as an aid in prognosis and, furthermore, can be used in treatment The entire apparatus consists of an obelisk-shaped wooden box 1 meter in length, at the narrow end of which a viewing tube is placed. In this tubelike opening the eyepiece (*A*) has a — 11.00 D. sphere permanently inserted. In front of this is a lens holder (*B*), movable from side to side, in which is firmly set (*1*) a — 2.00 D. sphere, used for viewing targets during the lesson period at a distance of 1 meter; (*2*) a — 6.00 D. sphere, used in viewing the test chart at 20 feet (6 meters), and (*3*) a blank space for insertion of a lens of any other power for investigative purposes.

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3. Mrs. S. B., aged 47, the wife of a physician, had complete suppression of the left eye and yet had normal vision with a + 1.25 D. sphere for each eye. Mrs. L. W., aged 27, had alternating suppression and yet had normal vision with a — 1.25 D. sphere for the right eye and a — 2.00 D. sphere for the left eye. Still another patient, Mrs. R. R., aged 35, had unilateral suppression of the right eye with normal vision with a correction of + 3.50 D. sph. + 2.25 D. cyl., axis 105; yet the left eye, with vision of 6/12 + with a correction of + 3.00 D. sph. + 2.25 D. cyl., axis 80, had no suppression. It is of interest to note that in case 19 (table 2) of the present series vision was 20/20 in the right eye and 20/300 in the left eye and yet there was no suppression in either eye. (These 4 patients were not included with the 50 patients discussed in the body of the paper.)

In the body of the eyepiece is a +6.00 D. spherical lens (*C*), which can be moved ½ to 3 inches (1.25 to 7.5 cm.) from the fixed —11.00 D. spherical lens. A scale (*a*) on the eyepiece is marked off in ½ inch (1.25 cm.) intervals, beginning at the fixed lens in the eyepiece. This scale gives the range of distance at which good focusing is obtained. A push button arrangement (*D*) on the holder of the lens (*C*) in the eyepiece housing moves the lens up and down. This stimulates the eye to overcome any existing suppression during the lesson.

At about the center of the device is the objective (*E*). Here is placed a +3.00 D. sphere actuated by a handle (*F*), effecting a movement from 6 to 14 inches (15 to 35 cm.), which represents the distance from the eyepiece to the objective.

At the extreme end of the device is a lamp-housing device (*G*), in which are placed two 8 watt, fluorescent bulbs of daylight quality. In the lamp housing also are four 25 watt lamps, two in red and two in green, with separate switches for each colored lamp. The colored lights can be used with the colored targets of contrasting colors for the attainment of fusion of color when this is desirable. When a target is not inserted at the back of the lamp housing, a view of distance (infinity) is obtained. This is how vision is taken through the apparatus at 20 feet with a Snellen card as a target. A trapdoor (*H*) at the side can be used to insert an indicator to point out to the patient which part of the target is under discussion. Without the target the subject looks out into space, and therefore the apparatus has telescopic qualifications. For this reason, and for want of a better name, the device is called a telescopic amblyoscope.

TARGETS

The targets are improvised by taking heavy cardboard (6¼ by 6¾ inches [15.8 to 17 cm.]) and pasting thereon pictures showing detail, which the subject is required to describe carefully. Other interesting charts are news clippings from microfilm size newspapers. Targets are also made which utilize the smallest (no. 1) type on the Jaeger test chart, as arranged by Dr. S. Lewis Ziegler, to letters 3 inches (7.5 cm.) in height.

If the lessons were given slowly, as they were, it was interesting to note that the fact that the patient remembered the targets was not a handicap. The letters on the target were then studied more carefully, and possibly an inherent inferiority complex was overcome, with resultant cooperation on the part of the patient.

TECHNIC OF USING THE TELESCOPIC AMBLYOSCOPE

The device may be used in one of two ways. With either method the —2.00 D. sphere (*B*, figure 1) is placed before the eyepiece if the reading target is inserted at the 1 meter distance, or the —6.00 D. sphere is used without targets for reading at 20 feet or more. The visual correction is worn by the patient.

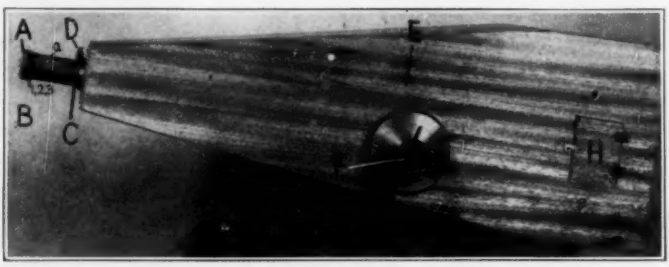
With method 1, the handle (*F*), which actuates the objective (*E*) is placed at *8* or *10*. This signifies a distance of 8 or 10 inches (20 to 25 cm.) from the eyepiece. The lever (*D*) operating the lens in the eyepiece is then moved from *0* to *1½* on the scale (*a*), denoting the distance in inches (1.5 to 3.75 cm.) from the movable lens (*C*) to the fixed lens (*A*), contained in the eyepiece. Occasionally while the patient is reading, he may note a blur. This indicates ocular suppression, which is improved by pressing several times quickly on *D*, which moves lens *C* up and down, thus overcoming the blurring. It is best to continue using targets of progressively smaller type. Photomicrographic newspapers, such as were sent overseas during the recent war, were found most satisfactory. (The topics were interesting, and the type was minute as seen at a distance of 1 meter.)

With method 2, the technic is the same as in the method just described except that the lever (*F*), after good vision is obtained by the patient, is moved back and forth to obtain first clear and then poor vision, an exercise to stimulate the eye to better vision by means of accurate focusing.

During the lesson a certain amount of time was devoted to overcoming any other visual obstacle present in addition to amblyopia. At the first evidence of fatigue during the visual training the lesion was stopped, and the rest of the lesson period was devoted to overcoming the obstacles present by treatment with the synoptophore, the cheiroscope, the rotoscope or any other device deemed advisable by the technician.

PLAN OF STUDY

At the first examination each patient had his vision taken on three types of test charts, usually the Snellen E chart for illiterate persons and the letter and number charts.[4] As is well known, certain letters are more easily read than others. Ordinarily, letters or numbers, as demonstrated in the "Manual of the Medical Department" by Lebensohn [5] are more difficult to discern. For example, an *8* looks more like *B; P,* or *T,* are misread for the letter *F,* and *D* looks like *B* or *O.* It was observed that discrimination is easier with the white letters on a black background.



Telescopic amblyoscope.

A, a —11.00 D. sphere permanently fixed; *B,* a sliding lens holder, containing (*1*) a —2.00 D. sphere, (*2*) an open space for inserting a lens of any diopter strength for experimental purposes and (*3*) a—6.00 D. sphere; *C,* + 6.00 D. sphere operated by holder *D,* in the center of which is a rod which moves lens *C,* and thus may be used to overcome suppression while giving the lesson; *a,* an engraved plate to give the exact distance of *C* from *A*; *D,* holder for the + 6.00 D. sphere (*C*), which effects its movement; *E,* + 3.00 D. sphere, the objective of the telescope; *F,* dial giving the distance of the objective from the ocular; *G,* target holder, and *H,* trapdoor used by the technician to view the target and indicate where the patient should fix his attention.

To make certain that the test charts were not memorized, a large variety of charts were utilized in checking the visual acuity.

As in orthoptic training, there was great difficulty in making appointments. Children, with few exceptions, could be seen only after school hours, from 3:30 to 5 p. m., and on Saturdays between 9 a. m. and 1 p. m. For adults the convenient time was the lunch hour, from 12 to 1 p. m., and the Saturday morning hours. It should be remembered that, as in orthoptic training, the best results are obtained in the early hours of the day, when the child is not fatigued. For

4. The illumination of the chart was 70 foot candles, evenly distributed over the entire chart.

5. Lebensohn, J. E.: Visual Rating, and Presentation of Improved Unlearnable Letter Chart, U. S. Nav. M. Bull. **41**:744, 1943.

this reason some of the patients were induced to come during school hours. In a few instances the teacher was cooperative in this venture and permitted the child's absence from school. The improvement in vision in these cases was gratifying.

Lessons were given once a week, lasting about one-half to three-quarters hour, depending on the cooperation of the patient. It was also found that, as in orthoptic treatment, a lesson lasting much longer fatigued the patient. In this group it was generally found impossible to get the patient to come oftener than once a week.

After the patient had been given twelve lessons (one a week), he was given a period of rest of one month. On the patient's return, after one month, the vision of both eyes was taken. This temporary interval between lessons was to make sure that the improved vision of the previous series of lessons was retained. If vision was not normal,[6] it was again taken through the telescopic amblyoscope with the Snellen test chart as a target at 20 feet (6 meters), just as was done before the first series of twelve lessons was given. If vision still showed possibilities of improvement, another series of twelve lessons was suggested. Any improvement, if obtained, came within these two series of lessons. The improvement obtained during a third series of twelve lessons was so slight that it hardly deserved the time and trouble taken. Unfortunately, a few patients included in this series who attained a 20/30 vision could not be persuaded to complete the course of treatment to attain 20/20 vision. Usually refusal was ascribed to illness in the family, distance to be traveled for lessons or inconvenience of time of appointment. However, these patients were asked to return regularly for a recheck of vision, which always remained constant. With few exceptions, all the patients who came for lessons kept their appointments regularly.

Ordinarily, one should see some improvement in vision at the end of the fifth to the seventh lesson. In some cases the rate of progress was accelerated at this time. Patients whose vision did not improve after the seventh lesson were advised to discontinue treatment, particularly if their attendance was irregular. Defects such as suppression, lack of simultaneous macular perception, poor fusion or stereopsis, were treated in conjunction with the amblyopia.

Home work was given to a few patients. This consisted in placing the patient 20 feet from a visual acuity chart and patching the good eye. Glasses were required to be worn. The subject stepped forward to the greatest distance at which the smallest type of the E chart for illiterates was just visible to him, then slowly stepped backward to see whether this line could be retained. When this line was mastered at 20 feet, an attempt was made to study the line of next smaller type on the test chart. The E or the broken ring chart is most suitable for this type of work, since by cutting the chart in half and turning it on its four sides eight different test charts are obtained. The individual chart in different positions is difficult to memorize. As stated previously in regard to targets, even if a patient remembers the letters of a chart, the success of the home training is not materially affected. To obviate fatigue, only five minutes of this home instruction should be given at a time. The exercise may be repeated after several hours. Continuous patching of the good eye was prescribed for a few patients, but it is questionable how adequately this treatment was carried out.

PROGNOSIS

The prognosis of amblyopia has always been difficult to make. Pugh [7] claimed that it was impossible to make a prognosis of the result

6. All patients' vision was taken with the glasses on.

7. Pugh, M. A.: Squint Training, London, Oxford University Press, 1936, p. 39.

of treatment in any case of amblyopia. It was found in a previous study with the Wollensak Allscope [1] that little could be expected by way of treatment from patients who did not have vision of 20/70 on the Snellen chart.

A patient whose vision was no better than 20/100 while looking through the telescopic amblyoscope was considered as possibly having congenital amblyopia. Such patients were not regarded as amenable to treatment. However, in spite of this, a few such patients were given visual training by way of experiment, with the expected poor result. Usually the initial vision taken through the telescopic amblyoscope with the Snellen chart at 20 feet was the final vision, plus or minus one line on the test chart, a change which one could expect the patient would show after his twelfth lesson with the wearing of glasses. This improvement occurs provided there is not absolute suppression. When there is pronounced suppression, it is best to attempt a correction of this condition before a definite prognosis is made. Often exercises given for one eye improved the sight of both eyes. This was also true of suppression in one eye. Improvement in vision was usually associated with improvement in suppression.

REVIEW OF CASES

The total series of patients taking treatments was 47. The ages varied from 6 to 46 years. Of the patients studied, those from 6 to 38 years of age showed the most promising results. Altogether, 56 patients were studied; 9, after preliminary studies were completed, could not return, for such reasons as inconvenience of clinic hours and illness; they therefore are not included in the present series.

Of the 47 patients, the majority, 34, or 72 per cent, ranged in age from 6 to 15 years, inclusive. It was interesting to note that in this small series there were 33 males and 14 females. Vision was defective in 14 right eyes and in 25 left eyes, and in 8 patients both eyes were ametropic. All the eyes, of course, had normal fundi. Twenty-five patients had anisometropia, all of whom obtained improvement in vision, except for 1 who showed no improvement whatever, and 2 who showed questionable improvement. Two patients had no refractive error.

Two trained technicians took care of the patients and interchanged patients in treatment, so that an error in judgment concerning the treatment of a particular patient would less likely be made. Five patients took two series of twelve lessons each, with a period of rest of four or five weeks between the two series. Two patients took a third series of lessons; like the preceding group, they had a rest period of four or five weeks before beginning each new series.

One patient, J. B., case 7, had improvement in vision but vision was extramacular, so that this patient, even though the vision improved from 20/200 to 20/40, could not be said to have improvement in vision.[8]

Unfortunately, the school vacation period came on before treatment of all the patients was completed, and 10 patients discontinued treatment. Of these 10 patients, 5 (cases 9, 11, 15, 40 and 47), showed definite improvement and were well on the way to emetropic vision. Five patients (cases 14, 21, 32, 43 and 45) did very poorly, possibly because of irregular attendance, and they were advised to discontinue the treatment. The oldest patients who showed improvement were M. L., (case 24), aged 40, and A. H. (case 22), aged 37 (table 1).

Suppression.—In the case of C. F. (4), the suppression became milder at the end of the second series of twelve lessons, and in the case of J. G. (6), the suppression entirely disappeared during the second course of lessons. In some of the patients examined suppression continued; yet vision improved in spite of this. For example, in cases 9, 15, 16, 29, 30 and 37 unilateral suppression changed to alternating suppression. Vision improved in all these cases. Often both the vision and the suppression improved to the same degree. In cases 16 and 37 vision improved more than the suppression.

In 3 patients with alternating suppression the results of treatment were as follows: F. S. (case 45) showed no improvement in vision or in suppression; R. E. (case 21) exhibited slight improvement in suppression with notable improvement in vision, and in J. K. (case 35) the alternating suppression became unilateral suppression with decided improvement in vision.

The other patients with alternating suppression (cases 10, 24, 25, 26, 31, 35 and 46) showed improvement in vision after taking lessons.

Fusion.—Thirty-seven patients had fusion before they came for treatment. One patient never obtained fusion. One patient obtained fusion at false projection. The remaining 8 patients obtained fusion in from two to eight lessons.

Stereopsis.—Twenty-nine patients had stereopsis before they began visual training. Of the remaining 18 in the series, only 10 patients had

8. A case similar to case 7, that of M. K., a woman aged 61, was not included in this series because of the patient's age, and particularly because of the ocular condition, which was such that little could be expected from any treatment. The patient had rotary nystagmus, right convergent squint of 45 degrees and palsy of the inferior oblique and superior rectus muscles. Vision was equal to light perception in the right eye and 6/300 in the left eye. After several irregularly taken lessons, vision in the left eye improved to 12/300. She also felt that the nystagmus was less. She was enthusiastic about her improvement in vision, saying that she could see the cracks in the sidewalk and that the curb was more clearly visible. Since she was so enthusiastic about the treatment, it was deemed unnecessary to explain to her that although the treatment was partially successful the improvement was not of the kind desired, that it might represent extramacular vision.

questionable stereopsis at the completion of training, and the other 8 obtained stereopsis in from five to twelve lessons.

Anisometropia.—There were 24 patients with anisometropia in this group. In 19 of these patients the condition improved. Three showed a questionable improvement, and 2 had no improvement whatever.

TABLE 1.—*Résumé of Clinical Data on Forty-Seven Patients Treated with the Telescopic Amblyoscope*

Case No.	Name	Sex	Vision Before Treatment	Vision After Treatment	Total No. of Lessons
1	L. A.	M	20/100	20/30	24
2	J. S.	M	20/50	20/30	34
3	W. S.	M	20/200	20/50—	24
4	O. F.	F	20/200	20/50	24
5	O. B.	M	20/100	20/40+	12
6	J. G.	M	20/70	20/20—	22
7	J. B.	M	20/200	20/40—	31
8	R. P.	M	20/200	20/40—	24
9	S. B.	M	20/40	20/20—	7
10	S. L.	M	20/70 20/70	20/40 20/40	12
11	H. M.	M	20/50+	20/30	7
12	E. L.	F	20/70	20/50—	9
13	W. E.	M	12/200	20/100	12
14	R. L.	M	10/200	19/200	7
15	P. L.	F	20/50	20/30	11
16	C. M.	M	20/40	20/30+	12
17	J. S.	M	20/100	20/50+	12
18	H. S.	M	20/70	20/30	12
19	B. E.	M	20/300	20/100+	7
20	J. C.	M	20/70	20/40—	12
21	R. E.	M	20/70 20/70	20/30— 20/30—	12
22	A. H.	M	20/70+	20/40+	12
23	A. K.	M	20/70	20/40+	12
24	M. L.	F	20/50— 20/50+	20/40 20/40	12
25	R. R.	M	20/100	20/50	12
26	L. M.	M	20/70 20/70	20/40 20/50+	12
27	H. Y.	M	20/100	20/40	12
28	W. S.	M	20/100	20/40—	12
29	W. S.	M	20/100	20/50	12
30	J. S.	F	20/70	20/50	12
31	B. K.	F	20/200	20/100	12
32	B. K.	M	20/100	20/200	10
33	T. P.	F	20/40	20/20—	12
34	J. A.	M	20/70	20/50+	12
35	J. R.	F	20/50+ 20/50	20/30+ 20/30+	12
36	J. K.	M	20/100	20/40+	12
37	G. J.	M	20/50 20/50	20/30 20/30	12
38	E. C.	F	20/50	20/30+	12
39	D. K.	M	20/70	20/20—	4
40	B. B.	F	20/50+	20/30—	5
41	I. E.	F	20/50	20/20—	12
42	R. M.	M	20/70	20/40	12
43	R. P.	F	20/200	20/148	8
44	A. C.	F	20/50—	20/40+	6
45	F. S.	M	20/70 20/70	20/100 20/70	12
46	B. G.	F	20/70+	20/40—	12
47	H. R.	M	20/70	20/40	5

OBSERVATIONS BY PATIENTS

Clinically, a few of the patients felt better subjectively and volunteered information, as follows:

L. A. (case 1), aged 15, claimed to have "greater visual comfort" after the first course of twelve lessons.

J. G. (case 6), aged 7, said he had better visual comfort and that the "spot" he could not see through when looking ahead was "no longer there."

S. L. (case 6), aged 9, with lateral nystagmus, saw the blackboard better in school, felt more comfortable and was told that his eyes did not "shake as much."

L. M., (case 26), aged 26, with lateral nystagmus, said that "the pain in the back of the head and the general discomfort disappeared during the first course of twelve lessons." He was told by several members of his family, one a physician, that the lateral movements of his eyes were not so frequent.

G. J. (case 37), aged 9, who always had headache, said he "feels better in school."

R. M. (case 42), aged 8, said that "the pains and funny feeling" in his head had disappeared.

TABLE 2.—*Detailed Clinical Data on Four Patients Treated with the Stereoscopic Amblyoscope*

Case No.; Name; Age; Sex	Vision	Lesson No.	Vision Before Treatment, Left Eye	Progress	Vision After Treatment, Left Eye
6†	Vision with correction:	1	20/70	Suppression	20/70
J. G.	R. E.: 20/30 with +3.25 sph. ⊃	2	20/60+	in left eye	20/50
7	+1.50 cyl., ax. 90	3	20/40—	noted	20/40—
M	L. E. 20/70 with +3.00 sph. ⊃	4	20/40—	throughout	20/40
	+1.50 cyl., ax. 105	5	20/40+	series;	20/40+
	Blindspot slightly enlarged on study of fields	6	20/30—	steropsis	20/40+
		7	20/30	obtained	20/40+
	Vision in L. E. 20/30 with stereoscopic amblyoscope at 6 meters	8	20/30	and kept	20/30+
		9	20/30+		20/30+
	Fusion present	10	20/30		20/30+
		11	20/30+		20/30
		12	20/30		20/30+
15‡	Vision with correction:	1	20/40—	Strong sup-	20/40
P. L.	R. E.: 20/30 with +5.50 sph. ⊃	2	20/40—	pression in	20/40
7	+0.25 cyl., ax. 90	3	20/40—	L. E.; fusion	20/30
F	L. E.: 20/50 with +6.00 sph. ⊃	4	20/30	obtained,	20/30
	+1.00 cyl., ax. 90	5	20/30+	suppression	20/30
	With amblyoscope at 6 meters vision in L. E. 20/30	6	20/30+	now alternat-	20/30+
		7	20/30+	ing and con-	20/30+
	S. M. P.¶ : fusion obtained on third lesson	8	20/30+	tinued as	20/30+
		9	20/30+	such with	20/30+
		10	20/30+	improvement	20/30—
		11	20/30+		20/20+
		12	20/30+		
19§	No refractive error present	1	20/300	No suppression	20/200
B. E.	R. E.: 20/30	2	20/300	noted;	20/200
16	L. E.: 20/30; vision of 20/100 obtained with stereoscopic amblyoscope	3	20/200	steropsis	20/200
M		4	20/200	very poor	20/200
		5	20/100—		20/100
	No suppression present in spite of high visual error	6	20/100—		20/100
		7	20/100		20/100+
	Fusion present				
	Attendance irregular, lessons discontinued				
43‖	Vision with correction:	1	20/200	Began with	20/200
R. P.	R. E.: 20/20 with +1.00 sph. ⊃	2	20/200	complete	20/148
46	+0.25 cyl., ax. 90	3	20/200	suppression	20/148
F	L. E.: 20/200 with +1.50 sph. ⊃	4	20/148	of left eye	20/148
	+5.00 cyl., ax. 90; add +2.25	5	20/140		20/100—
	Temporary fusion	6	20/200+		20/148
		7	20/200		20/100
		8	20/148		20/148

* A complete table of data for the entire series of patients treated will be furnished by the author on request.

† The patient took twenty-two lessons. Note the gradual improvement in vision with succeeding lessons. Suppression was present up to the seventeenth lesson.

‡ The patient had strong suppression, which changed to the alternating type. Vision improved in spite of this.

¶ S. M. P. indicates simultaneous macular perception.

§ The patient had vision of 20/300 but no suppression. Vision at the seventh lesson did not improve to better than 20/100, and treatment was discontinued.

‖ The patient, aged 46, had complete suppression, which improved. Vision did not improve, and treatment was discontinued after the eighth lesson. Presbyopic patients whose vision is checked on the telescopic amblyoscope at 20 feet (6 meters) get vision unobtainable with visual training. This was noted in 2 other patients. R. P. had vision of 20/30 at 20 feet (6 meters) with the device.

COMMENT

The question naturally asked is, "How can training help the vision?" Lancaster's explanation [9] for this improvement in visual acuity can be logically applied to this form of visual training, a method which is scientific and accepts acknowledged ophthalmologic data. He expressed the belief that it is not the primary retinal sensation that is improved; "it is the neglected but vitally important cerebral part of seeing that has been trained." Dobson's opinion [10] on the improvement of squint went further but can also be applied to the visual improvement obtained in treatment of ametropia. She expressed the belief that the improvement is explained by an arrest of development either in the macula lutea or in the occipital cortex, or in some intermediate area. The improvement obtained with the type of visual training reported here is compatible with the aforementioned theories. Whatever may be the explanations for the cause of success in ocular training, the fact is that visual training has often been successful in the hands of ophthalmologists. Travers [11] has used reading exercises to improve the visions of applicants for the air service with some success.

Although the present series is too small for statistical purposes, certain generalities can be made as to the value of visual training with the telescopic amblyoscope in cases of defective vision. An attempt will be made to follow up these patients from time to time to determine the permanency of the improvements obtained.

It was noted that although the improvement in vision was not startling it was usually consistent and ever increasing, with few exceptions, from lesson to lesson (table 2). Examination after an interval of four weeks or slightly more with no visual training following a course of twelve lessons showed that the improvement in vision after the series of treatments was usually retained.

Suppression was found to be commonly present in persons with refractive errors, but improvement in vision after training was often accompanied with disappearance of the suppresion. In 4 patients, however, the suppression never improved and vision did not improve either.

The magnification of the target and the fine focus adjustment of the apparatus probably help the patient in overcoming his timidity and should aid him psychologically in cooperation toward bettering his vision.

The visual correction originally obtained with glasses must continually be made use of.

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9. Lancaster, W. B.: Present Status of Eye Exercises for Improvement of Visual Functions, Tr. Am. Acad. Ophth. (1943) **48**:413, 1944.

10. Dobson, M.: Binocular Vision and the Modern Treatment of Squint, New York, Oxford University Press, 1933, p. 29.

11. Travers, T. a'B.: Distinction Between Amblyopia and Organic Defects of Vision, Tr. Ophth. Soc. Australia (1941) **3**:82, 1942.

Visual training prevents the embarrassment which children usually suffer when the good eye is continuously patched.

Whether the improvement in vision is proportional to the number and frequency of the lessons received is not possible to state at this time. During next winter's course of treatments an attempt will be made to give some cooperative patients three lessons a week in order to determine the relative benefit obtained.

Anisometropia was no more difficult to cure than the high hyperopias or myopias. Some patients with 20/40 vision were more difficult to train than patients with anisometropia.

SUMMARY

An instrument, the telescopic amblyoscope, which can be used for prognosis and for the training of defective vision in patients with ametropia of various intensities, is described.

Forty-seven patients with intractable ametropia were given reading lessons using the instrument. The technic of training for improvement of vision is described.

A prognosis is made by having the patient look through the telescopic amblyoscope at a test chart 20 feet (6 meters) away. Vision thus obtained, plus or minus one line, is the final vision to be expected after twelve lessons.

The reading lessons were given once a week for twelve weeks; then patients were given a rest period of four to five weeks to see whether the visual improvement after the last lesson was retained.

If after the rest period another examination gave promise of the vision being further improved, another series of twelve lessons was given. Little improvement was obtained after the second series of lessons.

The visual fields were found to be of neither diagnostic nor prognostic value.

The role of suppression in cases of amblyopia is discussed. Only in cases in which the suppression is severe (or absolute) should this defect be overcome before a prognosis for the amblyopia is given. Milder suppression may be overcome or even remain unimproved and yet the visual acuity may become improved.

The degree of the initial visual acuity is not necessarily an index to the prognosis in the case. Ametropia with vision of 20/40 may be more difficult to treat than anisometropia.

A table is presented showing the number of patients, the number of lessons given and the initial and terminal vision of each patient, as well as a table showing the refractive error and weekly progress in each of 4 cases.

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NATURE OF GLAUCOMA

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GLAUCOMA is a condition in which the intraocular pressure is intermittently or continuously higher than the optimum pressure which the organism has developed as a physiologic norm. The chief problem which this condition presents is the means by which the intermittent or continuous increase in intraocular pressure is brought about. Assuming that there is an acceptable theory which explains the manner in which the increase in pressure is brought about, how do the various clinical forms of glaucoma fit such a theory?

In a publication in 1938,[1] I presented a new concept of the maintenance of normal intraocular pressure and a new theory of the pathogenesis of chronic simple glaucoma. In the following discussion, I shall restate (1) the concept of the maintenance of normal intraocular pressure, and (2) the theory of the manner in which an increased intraocular pressure is brought about. I shall then attempt to show how the various forms of glaucoma fit such a theory.

MAINTENANCE OF NORMAL INTRAOCULAR PRESSURE

Normal intraocular pressure is maintained within narrow limits of variation. As presented in my previous publication, I conceive it to be maintained by means of a mechanism consisting of an effector organ within the eye and a central regulating apparatus. The latter consists of a center in the brain which regulates the activity of the effector organ through a neural and, possibly, a hormonal control. Such a mechanism must be assumed for the maintenance of each and every one of the physiologic norms within the organism, including the normal intraocular pressure. The manner of the working of this mechanism can perhaps be clarified.

Effector Organ.—In 1938 I said:

> The effector organ for the maintenance of the normal intraocular pressure . . . consists of . . . at least three units: (1) the capillary walls and the walls of Schlemm's canal, the passage of fluid through which is regulated by the electrolytic content of its layers; (2) the amount of circulating aqueous, and (3) the amount of circulating blood.[1]

1. Elwyn, H.: Pathogenesis of Chronic Simple Glaucoma, Arch. Ophth. **19**: 986 (June) 1938.

In the containing, practically inelastic sclerocorneal coat the intraocular pressure is maintained by the aqueous in the chambers of the eye and by the blood in the vessels of the uvea, especially those of the choroid. The amount of aqueous within the chambers and the amount of blood within the choroidal vessels are normally of a certain definite quantity, just sufficient to maintain the normal pressure. The volume of aqueous is kept within bounds by the balance between production and outflow, and such a balance is primitively regulated by the constellation of electrolytes in the separating membranes through which fluid (aqueous) moves into the posterior chamber and out through the angle of the anterior chamber and Schlemm's canal. Little is known about the regulating electrolytes; but, in analogy with other membranes of the body through which fluid moves, there is no doubt that such a primitive regulation exists. When, for any reason, the volume of aqueous increases or diminishes, the change of pressure on the transmitting membranes causes the additional central regulation to act in reestablishing the normal quantity.

Similarly, the volume of blood within the vessels of the choroid is of a definite quantity and is maintained within these vessels in just sufficient quantity to help, with the aqueous, in maintaining normal intraocular pressure. Here, also, any increase or decrease is immediately counterbalanced by the primitive regulating mechanism of electrolytes within the choroid, and more definitely by the central regulation. The result is the continuous maintenance of a definite volume of blood within the vessels of the choroid, which, with the constant volume of the aqueous, maintains the continuous normal intraocular pressure.

There are, then, in the effector organ for maintenance of the intraocular pressure three parts, or units; (1) the site of production of the aqueous, for the greater part the ciliary body; (2) the site of outflow of the aqueous, for the greater part the angle of the anterior chamber with Schlemm's canal, and (3) the blood vessels of the choroid. The importance of these three units in the maintenance of normal intraocular pressure is shown by certain pathologic conditions which involve separately each of these units.

1. The importance of the ciliary body in production of the necessary quantity of aqueous is shown in the lowering of the intraocular pressure when the ciliary body is diseased, as in inflammation, or when it is partly destroyed.

2. The importance of the angle of the anterior chamber in the outflow of the aqueous is shown by the sudden rise in the intraocular pressure when the angle is suddenly obstructed. Such an obstruction may be produced by a dislocated lens in the anterior chamber or by the filling of the angle with herniated vitreous, the latter being an occasional complication after extraction of a cataract within the capsule.

3. The importance of the choroidal vessels for maintenance of the intraocular pressure is shown by an experiment which nature occasionally performs. In cases of nevus flammeus of the face associated with glaucoma (part of the Sturge-Weber syndrome) there is an increase in the intraocular pressure. Before secondary changes, due to the increased intraocular pressure have occurred, there are no pathologic changes in the angle of the anterior chamber, in the ciliary body or anywhere in the eye except in the choroid. Here, there occurs a thickening due to an angioma, consisting of dilated spaces with very thin walls, which are lined by endothelium and filled with blood. The appearance is that of a cavernous angioma. In such cavernous spaces the constant volume of blood, which should not exceed that necessary to help maintain the intraocular pressure, cannot properly be regulated, and the result is an increase in intraocular pressure.

Central Regulation.—In 1938 I stated:

> Nothing is known of any center in the brain which controls the maintenance of intraocular pressure. In spite of this, it is necessary to hold to the conviction that there is such a controlling center. . . . [It] would probably be located in the brain stem, in the gray matter surrounding the canal system, . . . where so many of the centers for vegetative functions are located. The considerations which lead me to assume the existence of such a center . . . mainly are (1) the analogy with other vegetative functions which are known to be controlled by a center in the brain; (2) the inadequacy of the assumption that the action of purely physical forces alone and without regulation can explain the maintenance of such a physiologic constant as the normal intraocular pressure, and (3) the independence of the maintenance of the normal intraocular pressure from other bodily functions.

These considerations still hold good.

The Means of Control.—I have assumed, in analogy with other vegetative functions, that the means of control are hormonal and neural. For control by means of any hormone derived from any gland of internal secretion, there is at present no evidence whatever. There is evidence for control by means of nerves.

Neural Control.—I have said that the units in the effector organ for the maintenance of intraocular pressure are: (1) the angle of the anterior chamber, (2) the ciliary body and (3) the blood vessels of the choroid. These units receive their nerve supply from the parasympathetic ciliary ganglion, from which the short ciliary nerves leave as postganglionic fibers to enter the uvea. Their preganglionic fibers are contained in the oculomotor nerve and originate in the small-celled Edinger-Westphal nucleus, in the midbrain. The postganglionic fibers terminate in the tissues of the angle of the anterior chamber, in the ciliary body and in the choroid and its vessels. Convolutes of myelinated and nonmyelinated fibers are to be seen in the intervascular tissue of the choroid and the ciliary body. It can be assumed that these nerve fibers

serve the regulation of the functions of the choroidal vessels, of the ciliary body and of the tissues of the angle of the chamber. According to Eisler,[1a] the nerve supply of the choroid, including its vessels, is derived from the short ciliary nerves only.

The ciliary ganglion receives also a sympathetic root, which enters into relation with the parasympathetic fibers from the ciliary ganglion. It is possible that the eventual termination of the sympathetic fibers is as vasomotor fibers in the uvea. However, it is definitely established that vasodilator fibers for the eye run in the fifth nerve, according to Abderhalden,[2] who cited the literature on this subject. These fibers possibly run in the nasociliary nerve, which is a branch of the ophthalmic nerve, and from which a branch joins the ciliary ganglion as a long root. While the character of the nerve fibers in the sympathetic root of the ciliary ganglion and that of the fibers in the root derived from the nasociliary nerve are not definitely established, the character of the fibers which leave the ciliary ganglion in the short ciliary nerve as postganglionic fibers, and which originate in the cells of the ciliary ganglion, is definitely known. They are cholinergic and belong to the parasympathetic system. This points to a parasympathetic control of the functions of the choroid, of the ciliary body and of the angle of the anterior chamber, with its venous system. It also points to a parasympathetic control of the intraocular pressure.

MEDIATION OF ACETYLCHOLINE AND EFFECT OF PHYSOSTIGMINE

Further evidence that control of the intraocular pressure is by means of the cholinergic nerves of the parasympathetic system is given by the known action of physostigmine. The original studies of Otto Loewi and the work of all the investigators who followed in his steps have established that nerve fibers exercise their influence on effector cells by means of chemical mediators. It is known that the chemical mediator of the postganglionic fibers of the parasympathetic system is acetylcholine, which is produced at the nerve endings and which acts on the effector cells. It is known that acetylcholine is quickly destroyed by the specific enzyme cholinesterase, present in the blood and tissues of the body, which splits it into choline and acetic acid. The action of choline is weak compared with that of acetylcholine. Physostigmine in low concentration inactivates cholinesterase by combining with it, and in this manner permits the acetylcholine to act longer and more intensively.

1a. Eisler, P.: Die Anatomie des menschlichen Auges, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 1, p. 70.

2. Abderhalden, E.: Lehrbuch der physiologischen Chemie in dreissig Vorlesungen, ed. 1, Berlin, Urban & Schwarzenberg, 1925, vol. 2, p. 317.

Although acetylcholine is quickly destroyed in the tissues, the possibility of its being found within the eye has been thought of. Velhagen [3] demonstrated the "parasympathetic stuff" of Loewi, which is identical with acetylcholine, in the aqueous of certain animals. Engelhart [4] instilled physostigmine into the eye of rabbits to prevent the destruction by cholinesterase of any acetylcholine present and then exposed the eyes to light for parasympathetic stimulation. Under these conditions, he proved the presence of acetylcholine in the aqueous. Bloomfield [5] attempted to determine quantitatively the acetylcholine content of the aqueous of human eyes prepared according to the method applied by Engelhart to rabbit eyes. He tested 10 eyes with various noninflammatory pathologic conditions, but without glaucoma. The aqueous of each of the 10 eyes produced an inotropic depression of the heart beat. In 8 of the experiments the intensity of this effect was approximately equal to that produced on the same heart by a similar amount of acetylcholine in 1 : 100,000,000 dilution. In the remaining 2 essays the effect was one which could be reproduced with known concentrations of acetylcholine of over 1 :1,000,000.

These studies leave no doubt that stimulation of parasympathetic fibers of the eye leads to the production of acetylcholine in the eyes, which then overflows in the aqueous when its destruction is prevented by physostigmine.

In the early stages of chronic simple glaucoma, before any secondary organic changes have occurred in the eye, the intraocular pressure is increased at various times and is normal at others. In the period of increased intraocular pressure, the instillation of physostigmine into the conjunctival sac lowers the intraocular pressure to normal. With the known action of physostigmine, it is obvious that with inactivation of cholinesterase any acetylcholine present is permitted to act on the cells of the effector organ.

Here is definite evidence that the intraocular pressure is regulated by nerve impulses mediated by acetylcholine. The question is: Where do the nerve impulses originate? Obviously, not in the nerve endings or in the nerve fibers, for nerve impulses originate only in nerve cells. Do the nerve impulses for the regulation of intraocular pressure originate in the cells of the ciliary ganglion? Assuredly not! It has been pointed

3. Velhagen, K., Jr.: Zur Frage der vagotropen Substanzen im Auge, Arch. f. Augenh. **105**:573, 1932.

4. Engelhart, E.: Der humorale Wirkungsmechanismus der Oculomotoriusreizung, Arch. f. d. ges. Physiol. **227**:220, 1931.

5. Bloomfield, S.: Parasympathomimetic Effect of Aqueous Humor in Human Eyes With and Without Chronic Simple Glaucoma, Proc. Soc. Exper. Biol. & Med. **60**:293, 1945.

out by Gaskell[6] that the peripheral ganglions of the autonomic nervous system, to which the ciliary ganglion belongs, are intermediary relay stations, and purely distributive in character. They are definitely not regulative, but continue the propagation of impulses received from the preganglionic fibers. These preganglionic fibers are contained in the third nerve and are derived from the cells of the Edinger-Westphal nucleus. Impulses for the regulation of the intraocular pressure must, therefore, originate in the cells of the Edinger-Westphal nucleus, or in some center in the brain close by which sends impulses to the cells of the nucleus.

It is apparent from this discussion that there is evidence of a central regulation of the intraocular pressure and that such a regulation is effected by means of the parasympathetic fibers of the third nerve, mediated by acetylcholine, which acts on the cells and tissues of the effector organ in the eye.

SUMMARY

I conceive the mechanism for maintenance of normal intraocular pressure to be as follows:

1. There is a unit mechanism consisting of an effector organ within the eye and a central control from a center in the brain mediated by means of cholinergic nerves of the parasympathetic nervous system.

2. Normal intraocular pressure is maintained within the containing sclerocorneal coat by means of a constant definite volume of aqueous in the chambers of the eye and of a constant definite volume of blood in the vessels of the choroid.

3. The effector organ for the maintenance of intraocular pressure consists of three units: the tissues of the angle of the anterior chamber with Schlemm's canal, the ciliary body and the vessels of the choroid.

4. The constant volume of aqueous is maintained by the activity of the ciliary body and that of the angle of the anterior chamber, with its venous system. A primitive regulation is maintained in these tissues by means of a constellation of electrolytes. These tissues form one arm of the effector organ.

5. The volume of blood in the vessels of the choroid is regulated by the walls of the vessels. Here, also, a primitive regulation is maintained by means of electrolytes. These vessels form the second arm of the effector organ.

6. The primitive peripheral regulation is not sufficient in the highly developed warm-blooded animal. As in all vegetative functions in such animals, the primitive peripheral regulation is superseded by a central regulation.

6. Gaskell, W. H.: The Involuntary Nervous System, London, Longmans, Green & Co., 1920.

7. Central regulation is effected from a center in the brain, probably situated in the brain stem near or in the Edinger-Westphal nucleus.

8. From this center, impulses are propagated by way of the preganglionic fibers to the cells of the ciliary ganglion. Here they are redistributed and are propagated by means of the fibers of the short ciliary nerves to the tissues of the angle of the anterior chamber and its venous system, to the ciliary body and to the vessels of the choroid.

9. At their nerve endings the impulses are mediated by means of acetylcholine, which acts on the cells and tissues of the three units of the effector organ.

HOW IS THE INTERMITTENT OR CONTINUOUS INCREASE IN INTRAOCULAR PRESSURE BROUGHT ABOUT?

With the understanding of the mechanisms for the maintenance of normal intraocular pressure, the chief problem can be attacked: How is the intermittent or continuous increase in intraocular pressure brought about?

For the intraocular pressure to be continuously increased there must be an increase in the constant volume of the aqueous, or an increase in the constant volume of the blood in the vessels of the choroid, or an increase in both. Such an increase can be conceived to be produced (1) by organic changes and (2) by functional means.

1. Organic changes can produce an increase in the constant volume of the aqueous by interfering with its outflow. Such interference is produced by dislocation of the lens into the anterior chamber; by clotted blood or herniated vitreous filling the anterior chamber; by organic changes in the angle of the anterior chamber, such as follow obstruction of the central vein in some cases; by anterior synechias, such as occur in the later stages of glaucoma; by iris bombé and by a tumor or an exudate so situated as to obstruct the angle of the anterior chamber.

An increase in the constant volume of the blood in the choroidal vessels can be brought about by organic changes in the character of the vessels, such as an angiomatous malformation, or by any tumor, mass of blood or exudate which interferes with the outflow of blood from the choroidal vessels.

All these organic changes cause an increase in the intraocular pressure, which is usually referred to as secondary glaucoma.

2. An increase in the constant volume of the aqueous by functional means, in the absence of any organic changes in the eye, can be conceived to be produced by an increase in the production of the aqueous or by a diminution in its outflow. Such a change would mean a breakdown in the primitive electrolytic regulation of the effector organ. However, such a functional change, while it may occur for a moment, calls

at once for a counterbalancing influence by the central regulation over the neural pathways and the chemical mediator acetylcholine. In the presence of a normal central regulation, such a peripheral functional change which would result in an intermittent or a continuous increase in the intraocular pressure cannot be assumed.

Similarly, an increase in the volume of blood in the vessels of the choroid due to a peripheral functional change cannot be assumed to result in an intermittent or a continuous increase in intraocular pressure as long as the central regulatory apparatus is functionally intact.

Functional Change in Central Regulation.—In the absence of any organic changes in the eye, and with the units of the effector organ intact, an increase in intraocular pressure can be conceived as due to a functional change in regulation. Such a change would mean that the chemical mediator acetylcholine, which exerts its influence on the cells of the effector organ, is not produced in the continuous amounts necessary to maintain the continuous normal intraocular pressure. Instead, it would indicate that the production of acetylcholine is irregular, at times in normal amounts, at times in diminished amounts and at times in such small quantities that the intraocular pressure would remain continuously high.

Three possibilities present themselves: 1. A functional change at the nerve endings, where the acetylcholine is produced. This possibility is not acceptable, for any diminution in acetylcholine must at once call into activity the regulating center. 2. A functional change in the ciliary ganglion. This possibility, also, is not acceptable as long as the regulating center is intact, for the ciliary ganglion is only a relay station for the impulses coming from the preganglionic fibers and the center of regulation. 3. A functional change in the center of regulation. This is an acceptable possibility and to my mind represents the one way in which a functional change in the central regulation can influence the peripheral effector organ. Such a center can be conceived as undergoing a functional change as a result of inherited characteristics, a herodoconstitutional deficiency. This is in analogy with other functional constitutional deficiencies of centers in the central nervous system.

The manner in which such a functional change in the center of regulation causes an increase in the intraocular pressure can be conceived as follows: Impulses from the eye reach the center, but the ability of the cells of the center to react to such impulses is diminished to a variable degree. Fewer impulses leave the center to reach the terminal nerve endings within the eye. The result is a diminution in the production of acetylcholine in the effector organ. Such a diminution of impulses from the center may be more pronounced in the arm of the effector organ, which has to do with the maintenance of a constant volume of aqueous, namely, the ciliary body and the tissues of the angle of the anterior

chamber, with its venous system. Or, the diminution of central impulses may be predominant in the choroidal vessels, which have to do with maintenance of the constant volume of blood. In the first case, the constant volume of aqueous is not regulated centrally and is increased; in the second case, the constant volume of blood in the vessels of the choroid is not regulated and is increased. Or, all the effector units may receive a diminished number of impulses, and both the volume of aqueous and the volume of blood in the choroidal vessels are increased, and with this increase there is a corresponding increase in the intraocular pressure.

Several variations in increase in the intraocular pressure may be conceived:

1. There may be a slow and gradual reduction in the impulses from the regulating center, especially to units of the effector organ, which maintain the volume of the aqueous. The result is a slow and gradual increase in the intraocular pressure, with an increase in the volume of the aqueous.

2. There may be a slow and gradual increase in the volume of the blood in the choroidal vessels, with a resulting gradual increase in the intraocular pressure.

3. With the slow and gradual increase in the intraocular pressure, there may be sudden temporary extreme reductions in, or perhaps even complete cessation of, central impulses. The result is a sudden or sharp rise in the intraocular pressure. Such a sudden rise is especially likely to occur when there is a sudden increase in the volume of blood in the choroidal vessels.

That there is an actual reduction in the production of acetylcholine in the eyes with glaucoma may be inferred from the work of Bloomfield.[5] The aqueous of 7 eyes with chronic simple glaucoma was tested in a manner similar to that used with the aqueous of the 10 nonglaucomatous eyes previously mentioned. In no instance did a sample from these eyes produce a detectable parasympathicomimetic depression of the cardiac contraction. Bloomfield concluded that, in contrast to the aqueous of nonglaucomatous eyes, no acetylcholine-like substance is demonstrable in the aqueous of eyes with chronic simple glaucoma when tested by his method.

PATHOGENESIS OF GLAUCOMA

With the theory advanced for the maintenance of normal intraocular pressure, I have attempted to explain the manner in which an increase in the intraocular pressure can be brought about. Assuming the explanation to be acceptable, one may consider how the clinical forms of glaucoma fit it.

CLASSIFICATION OF GLAUCOMA

There are two fundamental types of glaucoma.

1. Secondary glaucoma, in which the increase in intraocular pressure is due to organic changes which affect the units of the effector organ for the maintenance of intraocular pressure in the eye.

2. Primary glaucoma, in which the increase in the intraocular pressure is purely functional in origin and is not due to any organic change in the eye. Organic changes occur in the later stages and are the consequences of the continuous increase in intraocular pressure.

Primary Glaucoma—Although a number of syndromes have been classified under this heading, there is in reality only one disease, primary glaucoma. Either the disease manifests itself by a slow, insidious increase in the intraocular pressure, and is then spoken of as chronic simple glaucoma, or its insidious course is interrupted by a sudden increase in the intraocular pressure, an acute attack, which, because the vessels of the eye are overfilled with blood, is spoken of as acute congestive glaucoma. Such an acute attack is not a separate disease, but only an acute episode in the course of primary or chronic simple glaucoma. The acute episodes are not severe at times; and, because of the milder manifestations of the individual symptoms, they are spoken of as prodromal attacks, meaning that they frequently precede a severe acute attack.

Primary glaucoma is thus one disease and manifests itself as (1) chronic simple glaucoma and (2) acute congestive glaucoma.

How does chronic simple glaucoma fit the explanation for the production of increased intraocular pressure which I have presented?

Chronic Simple Glaucoma.—The symptoms of chronic simple glaucoma are well known and need not be reviewed here. However, it is necessary to separate sharply the changes which are the consequences of the persistently high intraocular pressure from the symptoms which appear early in the course of the disease. In the early stages, before secondary changes have occurred, there are no visible alterations in the eye, either in the anterior segment or in the optic disk; and the eye does not differ in appearance from the normal eye. There are no subjective symptoms. At this time the eye with chronic simple glaucoma differs from the normal eye, first, in the greater lability or instability of the intraocular pressure during the twenty-four hours of the day, even when the low readings are still within the normal limits; and, second, in the gradual increase of the intraocular pressure above that for the normal eye.

The course is somewhat as follows: At about the age of 40, or shortly before, the intraocular pressure becomes more labile and shows greater variations. Tests made at this time show greater variations in response than in a normal eye. This lability or instability persists, and

within an indefinite period the intraocular pressure not only shows the greater variations but continues to be higher than that of the normal eye. Again, in an indefinite period the intraocular pressure becomes still higher, and with this persisting secondary changes begin to appear.

The absence of any organic changes in the eye in the early stages of this disease, and the whole course until secondary changes occur, leave no doubt that the disease is functional. As the transition from the normal intraocular pressure to a persistently increased pressure is a gradual one, and as the earliest indication of the disease is a greater lability of the intraocular pressure, a number of tests have been introduced which test this lability. Recently Bloomfield and Lambert [7] have developed a new test which seems to be an improvement over the older ones. A sphygmomanometer cuff, which has been placed loosely about the patient's neck, is inflated to a pressure of 40 to 50 mm. of mercury, while at the same time one of his hands is immersed over the wrist in ice water. With this test, which they called a lability test, Bloomfield and Lambert found that normal eyes reacted with an increase in the intraocular pressure of 1 to 9 mm. of mercury. The patients with the earliest stages of glaucoma reacted with an increase of 9 to 19 mm., and the patients with the more advanced disease, with an increase of 8 to 30 mm.

Bloomfield and Lambert also investigated the effect of drugs on the response to their lability test. One fact was brought out which is of great interest here. They found that pilocarpine in clinical dosage appreciably reduced the abnormal lability of the intraocular pressure in each of 13 eyes with chronic simple glaucoma on which operation had not been performed. I pointed out the importance of this observation in the discussion following their presentation.

Pilocarpine is a cholinergic drug and is known to act on the effector cells innervated by postganglionic cholinergic nerve fibers. The action is a direct one and occurs after nerve section and complete nerve degeneration. The drug has an effect similar to the muscarinic action of acetylcholine. The observation of Bloomfield and Lambert shows that pilocarpine administered to their patients with glaucoma acted on the cells of the effector organ for the maintenance of intraocular pressure in the same manner as does acetylcholine. The inference is clear: The effector organ is intact, for it responds to the action of pilocarpine, as it normally does to the action of acetylcholine. It can be inferred, further, that in eyes with chronic simple glaucoma acetylcholine is not produced in sufficient amounts to act continuously on the effector organ. Variations in the continuous state of the intraocular pressure can be assumed to be due to

7. Bloomfield, S., and Lambert, R. K.: The Lability of Ocular Tension: A Test to Determine Individual Variations, Arch. Ophth. **34**:83 (Aug.) 1945.

variations in the amount of acetylcholine produced at the nerve endings of the postganglionic fibers in the effector organ.

Primary Disturbance in Chronic Simple Glaucoma.—In the second section of the paper, I have given the reasons that a primary functional change in the postganglionic fibers or in the cells of the ciliary ganglion cannot be accepted. The only functional change which can be assumed is in the origin of the impulses in the regulating center in the brain. I come, then, to the necessary conclusion that the primary disturbance in chronic simple or primary glaucoma is a persistent functional deficiency in the central regulation for maintenance of the intraocular pressure, and that this functional deficiency is in the center in the brain from which the regulating impulses emanate.

There are no known etiologic factors but heredity which can be held responsible for such a deficiency in the central regulation for maintenance of the intraocular pressure. I have already discussed this in my previous publication.

As in my publication of 1938, I come to the following conclusion as to the nature of chronic simple or primary glaucoma:

The disease is a heredoconstitutional one, affecting the mechanism for the maintenance of normal intraocular pressure. In certain persons, as a result of an inherited defect, this mechanism is constitutionally inferior. This constitutional inferiority is already inherent in the germ plasm and is the cause of the comparatively shorter period of normal activity of the mechanism. In advancing life in most cases of glaucoma, or in early life in the rare cases of juvenile glaucoma, the mechanism loses its normal stability and is at first able to maintain normal intraocular pressure only in an incomplete manner. This is shown by the greater diurnal variations in the intraocular pressure and by the abnormal responses to tests. Gradually the inability to maintain normal intraocular pressure increases until it cannot be maintained at all. There is a functional change in the central regulation, by inference situated where the impulses for maintaining the normal intraocular pressure originate—in the center in the brain. I have said that the center of regulation is situated in or near the Edinger-Westphal nucleus. I am inclined to the assumption that the center is situated in this nucleus. Each half of such a bilateral paired center is subject to the same heredoconstitutional defects as the other half, but not necessarily to the same or to an equal degree. A bilateral paired center so situated explains certain variations in the course of primary glaucoma: (1) the difference in time of onset of the disease in the two eyes; (2) any difference in the tempo of progress of the disease in the two eyes; (3) the onset of an acute attack in the course of the disease in one eye only, or first in one eye and later in the other.

Variations in the Clinical Picture of Chronic Simple Glaucoma.—With the theory thus elaborated, there fit in certain variations in the clinical picture of chronic simple glaucoma. Usually in this disease it is found that the lens-iris diaphragm is pushed forward, with consequent diminution in the depth of the anterior chamber. In many cases, however, the anterior chamber is of normal depth, and ophthalmologists since Raeder frequently make the distinction between glaucoma with a shallow chamber and glaucoma with a deep chamber. Such a variation may be explained as follows:

The deficiency in the center of regulation causes a reduction in the number of regulating impulses to reach the units of the effective organ which maintain, respectively, the constant volume of the aqueous and the constant volume of blood in the choroidal vessels. The units form two distinct arms of the effector organ. With the deficiency in the center of regulation, lack of sufficient impulses must be assumed to affect both arms of the effector organ. But it is also conceivable that the lack of regulating impulses affects predominantly one or the other. When the units for maintaining the constant volume of the aqueous are predominantly affected, there will be predominantly an increase in the constant volume of the aqueous. The aqueous fills the chambers equally, and the anterior chamber remains normally deep.

When the lack of regulating impulses affects predominantly the unit of the effector organ for maintaining the constant volume of blood in the choroidal vessels, an increase in the intraocular pressure is predominantly due to the increase in the constant volume of the blood in the choroidal vessels. The result is a swelling of the choroid sufficient to push the vitreous and the lens-iris diaphragm forward, and the anterior chamber becomes shallow. As cases of glaucoma with a shallow chamber predominate over those with a normal or deep chamber, I assume that in most cases the constant volume of blood in the choroidal vessels is predominantly affected. This is perhaps borne out by the lability test of Bloomfield and Lambert. It will be recalled that this test consists of two parts: (1) immersion of the hand and wrist in ice water, causing a reflex increase in blood pressure, which drives an additional slight amount of blood into the vessels of the eye, and (2) constriction of the veins of the neck, which obstructs the outflow of blood from the vessels of the eye. The test is thus primarily a test to determine whether the eye will counteract an attempt to increase the constant volume of blood in the vessels of the choroid.

Acute Congestive Glaucoma.—An attack of acute congestive glaucoma is accompanied with (1) severe pain in the eye and in the head, usually in the distribution of the ophthalmic branch of the fifth nerve; (2) sudden reduction in vision; (3) ciliary injection, a cloudy cornea, a shallow anterior chamber, an immobile, dilated and frequently irregular

pupil and a plasmoid aqueous, and (4) a sudden rise in the intraocular pressure, which may reach 90 mm. or more. There is no doubt that all the ocular symptoms are due to the sudden increase in intraocular pressure, which causes a sudden tenseness and stretching of the corneoscleral coat and a sudden interference with the circulating blood in the vessels of the eye. It is the suddenness of the increase in the intraocular pressure that is important. This is the primary change, and it is necessary therefore, to explain this sudden increase.

It should be remembered that a sudden attack of acute congestive glaucoma is not a disease in itself, but that it is an acute episode in the course of chronic primary glaucoma. Whenever it occurs, even as the first indication of glaucoma, chronic primary glaucoma has already existed for some time. The same statement holds true for the milder prodromal attacks. The deficiency of the central regulation has already existed for some time. The assumption is therefore justified (1) that there occurs a sudden temporary reduction in, or perhaps even cessation of, the impulses from the central regulation and (2) that, as a result, it is the constant volume of blood in the vessels of the choroid that is more markedly and more quickly affected. The sudden pronounced increase in the volume of the choroidal vessel results in the sudden pronounced increase in the intraocular pressure, which is the cause of of the symptoms enumerated in the preceding paragraph.

That such a sudden reduction in the regulating impulses is not permanent is shown by the fact that an acute attack at times subsides by itself and by the fact that physostigimine at times produces a reduction of the acute attack.

SECONDARY GLAUCOMA

It is not necessary to review here the nature of secondary glaucoma. This is clear from the definition given earlier in the paper. In every case of secondary glaucoma there are organic changes within the eye which interfere with the functions of the units of the effector organ: (1) with the outflow of aqueous through the angle of the anterior chamber and its venous system, (2) with the circulation of the aqueous from the posterior to the anterior chamber and (3) with the maintenance of the constant volume of blood in the vessels of the choroid.

I have already mentioned the glaucoma which occurs with a nevus flammeus of the face, part of the Sturge-Weber syndrome. This form of secondary glaucoma is of great importance theoretically. The course of the disease imitates that of chronic simple glaucoma. Yet it is known that the glaucoma associated with this syndrome is secondary to an angioma of the choroid, evidence of the importance of the constant volume of blood in the vessels of the choroid.

CONCLUSION

With the understanding of the maintenance of normal intraocular pressure as outlined, the nature of primary glaucoma is conceived of as follows:

1. It is a heredoconstitutional functional disease, involving primarily the function of central regulation of the intraocular pressure.

2. In some persons, as a result of inherited influences, at about middle age or earlier the central regulation becomes unstable. Impulses from the center reach the effector organ in the eye in normal numbers at times and in smaller numbers at others.

3. As a result, the chemical mediator acetylcholine is produced in an irregular manner at the nerve endings in the effector organ, and the action of this organ is irregular. The intraocular pressure is normal at times and high at others.

4. The normal intraocular pressure is maintained by the constant volume of the aqueous in the chambers of the eye and by the constant volume of blood in the choroidal vessels. These constant volumes are maintained by the activity of the units of the effector organ in the eye.

5. The effector organ for maintenance of the normal intraocular pressure consists of three units: the ciliary body and the angle of the anterior chamber with its venous system, which maintain the constant volume of aqueous, and the choroidal vessels, which maintain a constant volume of blood in the choroid.

6. When insufficient impulses from the central regulation reach the units of the effector organ, either the constant volume of the aqueous is increased or the constant volume of the blood in the choroidal vessels is increased, or both are increased. The result is glaucoma with a normal anterior chamber when the increase in the volume of the aqueous predominates, or glaucoma with a shallow chamber when an increase in the constant volume of blood in the choroidal vessels predominates.

7. Primary glaucoma is one disease only. It either manifests itself by an insidious onset and a slowly progressive course—chronic simple glaucoma—or the course is at times interrupted by a sudden, intense increase in the intraocular pressure, which produces severe symptoms, characterized as acute congestive glaucoma, or milder symptoms, characterized as a prodromal attack of acute glaucoma.

8. Secondary glaucoma is the increase in intraocular pressure which results when the outflow of aqueous from the chambers of the eye or the constant volume of blood in the vessels of the choroid is interfered with by organic changes within the eye.

239 Central Park West (24).

CONTACT LENSES FOR CORRECTION OF MYOPIA COMPLICATED BY LESIONS OF THE RETINA AND CHOROID

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LOS ANGELES

THE ADVANTAGE derived from the use of contact lenses for the correction of refractive defects of the eye, as well as their value in the compensation of certain pathologic conditions of the cornea, has been extensively discussed in the literature. Rugg-Gunn [1] described the improvement in visual acuity obtained with contact lenses in different types of ametropia; O'Rourke [2] and Sitchevska [3] reported on the optical correction of keratoconus by means of contact glasses, and Cogan [4] demonstrated their value in cases of bullous keratitis. But little experience seems as yet to exist concerning the indication of contact lenses for compensation of visual impairment due to myopia concomitant with degenerative processes in the fundus of the eye. As far as could be ascertained, such a use of contact lenses has been reported only by Mihalyhegyi,[5] who in a series of 7 cases found contact lenses to be superior to conventional spectacles in the correction of the refractive error, as well as in the resulting visual acuity.

It is obvious that contact glasses are unable to improve degenerative conditions in the fundus of the eye. However, they are effective in the correction of faulty refraction, and increase the visual perception in myopic patients to a considerable degree. This fact is especially gratifying in cases in which myopia coincides with extensive pathologic changes in the fundus.

The beneficial effect of contact lenses will be appreciated when their specific advantages are clearly understood. With spectacles, the fixed

1. Rugg-Gunn, A.: Contact Glasses for Ametropia, Lancet **2**:1067 (Nov. 15) 1930.

2. O'Rourke, D. H.: Optical Correction of Conical Cornea with Contact Glasses, Am. J. Ophth. **12**:187 (March) 1929.

3. Sitchevska, O.: Contact Glasses in Keratoconus and in Ametropia, Am. J. Ophth. **15**:1028 (Nov.) 1932.

4. Cogan, G.: Bullous Keratitis, Arch. Ophth. **25**:941 (June) 1941.

5. Mihalyhegyi, G.: Improvement of Visual Acuity with Contact Glasses in the Presence of Lesions of the Retina and Choroid, Klin. Monatsbl. f. Augenh. **108**:200 (March-April) 1942.

lens and the movable eyeball are combined into a heterogenous optical system. This discrepancy, as Duke-Elder [6] pointed out, is apt to produce a great number of visual disturbances: The retinal image may undergo variations as to size; marginal changes in power are apt to produce distortions; oblique rays are not sharply focused on the retina but, rather, spread over a more or less diffuse area. Moreover, there

TABLE 1.—*Size of Retinal Image Depending on the Use of Contact Lenses*

Diopter strength of lens	—20	—10	—6	—2	0*	+2	+6	+10
Size of retinal image	1.455	1.19	1.10	1.03	1.00	0.97	0.92	0.865

* 0 represents ametropia, with the size of the retinal image indicated at 1.

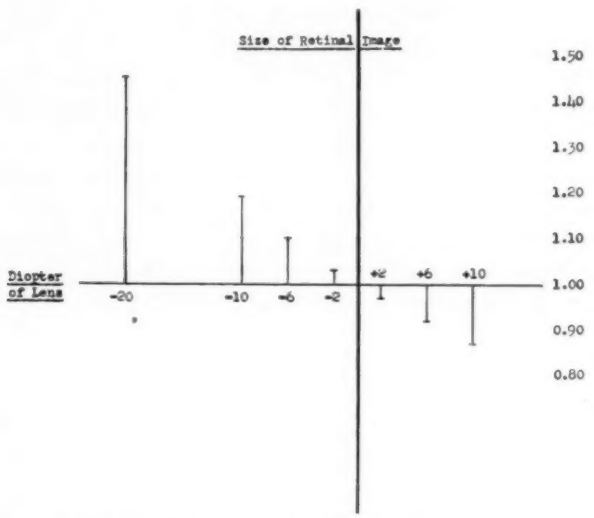

Fig. 1.—Size of retinal image, depending on the use of contact lenses.

may occur changes in perspective, chromatic aberrations and reductions in the field of vision. Occasionally, the use of spectacles may, furthermore, produce imbalance of the ocular muscles.

When contact glasses are prescribed, many of these disturbances will disappear. As the lenses are worn on the eyeball in apposition to the cornea, the distance between the lens and the eye is reduced to a minimum. The contact lens is centered in front of the pupil and moves in coordination with the eyeball, thus eliminating distortion, increasing the field of vision and preventing aberrations. The resulting gain in visual acuity assumes great importance, especially when myopia is com-

6. Duke-Elder, S.: Practice of Refraction, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1938, p. 337.

plicated by a pathologic condition of the fundus. Even in those cases in which through correction with conventional glasses only a slight improvement of vision can be achieved, contact lenses may lead to a noticeable increase in the accuracy of optical perception and afford the patient great relief.

The closer a concave lens can be brought to the eye, the larger is the resulting image. Thus, the greatest improvement in vision will be

TABLE 2.—*Data on Twelve Cases of Myopia Concomitant with Lesions of the Fundus in Which Contact Lenses Were Fitted*

Case	Age, Yr.	Pathologic Condition of Fundus	Vision with Glasses	Vision with Contact Lenses	Wearing Time, Hr.
1	17	Choroiditis (congenital syphilis)...	O. D.: 5/200 O. S.: counting of fingers at 2 ft.	O. D.: 20/40— O. S.: 20/100	7
2	24	O. D.: Retinochoroiditis ..... O. S.: Injury loss of eye	O. D.: 20/200	O. D.: 20/30	12
3	16	Destruction of choroidal and retinal tissues; peripapillary atrophy	O. D.: 20/40 O. S.: counting of fingers at 1 ft.	O. D.: 20/30 O. S.: 20/200	8
4	35	Retinitis pigmentosa	O. D.: 20/50 O. S.: 20/200	O. D.: 20/40 O. S.: 20/70	3
5	41	Small white choroidal patch near macula with pigmentary deposits	O. D.: 20/40— O. S.: 20/40—	O. D.: 20/20— O. S.: 20/20—	5
6	31	Choroiditis with extreme thinning of retina	O. D.: 20/60— O. S.: 20/60—	O. D.: 20/50 O. S.: 20/50	8
7	27	Grayish spots in macular areas...	O. D.: 20/40— O. S.: 20/40—	O. D.: 20/20— O. S.: 20/20	5
8	33	Choroiditis with heavy pigmentation and atrophic areas	O. D.: 20/50— O. S.: 20/50	O. D.: 20/30 O. S.: 20/30	7
9	32	Atrophic patches of retina and choroid with pigmentation	O. D.: 20/70 O. S.: 20/70	O. D.: 20/30 O. S.: 20/30	4
10	21	O.D.: Diffuse choroiditis..... O. S.: Normal	O. D.: 20/70— O. S.: 20/20—	O. D.: 20/50 O. S.: 20/20+	10
11	22	Small whitish yellow spots (Tay's choroiditis)	O. D.: 20/30 O. S.: 20/30	O. D.: 20/20 O. S.: 20/20	9
12	24	Choroiditis with atrophic areas around disks	O. D.: 20/30— O. S.: 20/40—	O. D.: 20/20— O. S.: 20/30	8

obtained when the lens is placed in almost physical contact with the cornea. On the basis of mathematical calculations, the increase in the size of the retinal image is approximately 20 per cent in the case of a — 10 D. lens. In carefully selected cases of near sightedness, therefore, patients will experience a considerable gain in visual acuity through the use of contact lenses.

In spite of all these objective advantages, the subjective element of the patient's psychology cannot be excluded. The prescription of

contact lenses will prove successful only if the patient fully realizes that in his particular case the wearing of such glasses is indispensable for obtaining adequate vision. My experience confirms the opinion of Thier [7] that for the patient the strong stimulus of poor vision is needed as a compelling incentive to induce him to use contact lenses, and to wear them progressively oftener and over a more extended period of time.

The present study is based on a series of 30 cases of myopia in which the patients were fitted with contact lenses. While in 18 of these cases there occurred only minor changes of the fundus, in 12 the eyeground presented evidence of a serious pathologic process. Visual acuity increased in all 30 cases, but the improvement was most pronounced in the cases with advanced degeneration of the fundus of the eye. The wearing time varied between a minimum of three hours and a maximum of twelve hours daily and amounted to an average of seven hours. There was no complaint of discomfort resulting from the use of contact lenses, but in a number of cases cloudiness and visual disturbances developed after varying periods of time, necessitating removal of the lenses. Some patients, however, were able to reinsert the lenses after thirty minutes to one hour and again obtain clear vision.

The nature and concentration of the "buffer solution" may have a certain influence on the patient's tolerance of the contact lens. But in at least 6 cases it was noted that the properties of the fluid were of no importance, and 1 patient, who wears his lenses for twelve hours every day, even uses plain water and asserts that it causes less disturbance and no irritation.

Thier [7] has shown in interesting experiments that whenever the contact lens does not fit too tightly the liquid is replaced by lacrimal secretion within fifteen or twenty minutes. It would seem, therefore, that the quality of the fluid used is of importance only in those cases in which the lens fits tightly over the eyeball and the lacrimal circulation cannot replace the solution quickly.

In the following section, 4 characteristic cases are described in which contact lenses led to improvement of vision in patients with degenerative conditions of the fundus of the eye, concomitant in every instance with myopia. The pathologic conditions included retinosis, choroiditis, retinitis pigmentosa and Jensen's retinitis (retinochoroiditis juxtapapillaris). In each of these cases all refracting media were clear and neither palpebral nor corneal lesions were encountered; in some instances opacities were present in the vitreous.

7. Thier, P. F. X., in Ridley, F., and Sorsby, A.: Modern Trends in Ophthalmology, New York, Paul B. Hoeber, Inc., 1940, p. 308.

REPORT OF FOUR CASES

CASE 1.—M. D., a girl aged 17, had congenital syphilis, which persisted in spite of active treatment over an extended period. The last Wassermann reaction of the blood was 3 plus.

Examination of the right eye with a —11.75 D. sphere revealed vision of 5/200; vision in the left eye with a —12.00 D. sphere permitted counting of fingers at a distance of about 2 feet (60 cm.).

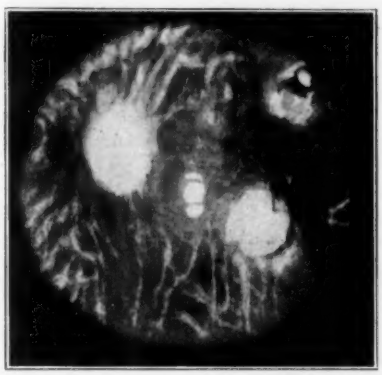

Fig. 2 (case 1).—Photograph of the left fundus, showing choroiditis concomitant with retinosis and pigmentation.

Fig. 3 (case 2).—Photograph of the right fundus. In the neighborhood of the macula, choroid, as well as retina, has been absorbed; a broad staphyloma separates the optic nerve from the retina.

Ophthalmoscopic studies demonstrated numerous floating opacities in the vitreous of both eyes. In the left eye choroiditis was encountered, coinciding with retinitis pigmentosa. As the retina was greatly thinned out, the choroidal landmarks had become clearly visible. Examination of the right eye led to similar findings.

Contact lenses were prescribed, and the patient is gradually becoming accustomed to their use. At present, she is able to wear them for a period of seven hours daily.

With contact glasses, vision has improved to 20/40 — in the right eye and to 20/100 in the left eye.

CASE 2.—G. G., a man aged 24, one year previously had lost the left eye as the result of injury. The best correction for the remaining (right) eye, of —14.00 sph. ⊃ —1.00 D. cyl., axis 180, gave vision of 20/200.

Inspection of the fundus of the eye revealed pronounced myopic changes. In its temporal portion, the optic nerve had become separated from the retina by a broad staphyloma. In the neighborhood of the macula appeared areas in which the choroid, as well as the retina, had deteriorated and the sclera was clearly visible. Throughout the entire fundus, the retina was rarefied and thinned to such an extent that the choroidal landmarks were pronounced.

A contact lens was prescribed, and the patient is able to wear it without any discomfort throughout the day. His vision has improved to 20/30. This improvement removed the obstacle of reduced eyesight and allowed the patient to return to his former work as a mechanic.

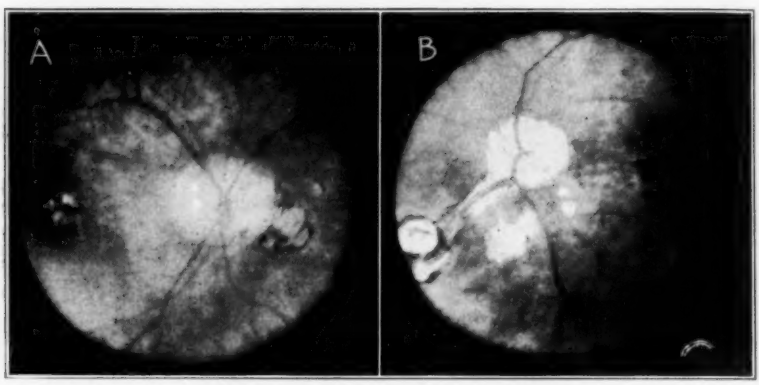

Fig. 4 (case 3).—*A*, photograph of the right fundus, showing complete destruction of the choroid in the region of the macula and in another area just nasal to the disk; *B*, photograph of the left fundus, showing areas of degeneration of the retina and choroid.

CASE 3.—R. B., a youth aged 16, had vision of 20/40 in the right eye with a correction of —7.00 D. sph. ⊃ —2.25 D. cyl., axis 180; vision in the left eye with a —7.00 D. sphere permitted counting of fingers at a distance of 1 foot (30 cm.).

In the right eye (fig. 4 *A*) an area of destruction of choroidal and retinal tissue and of pigmentation was present on the nasal side, starting from the optic disk. Around the papilla atrophy was clearly evident. Rarefaction of the retina had progressed to such a point that the choroidal landmarks had become visible. The region of the macula was marked by complete destruction of the choroid and was patterned with deposits of pigment. In the left eye (fig. 4 *B*) the degenerative condition of the retina, accompanied with heavy pigmentation, was clearly evident. An area of choroidal atrophy appeared below the disk in the nasal direction.

Contact lenses were prescribed, which the patient is wearing daily for a period of at least eight hours. With these glasses vision has improved to 20/30 in the right eye and to 20/200 in the left eye.

CASE 4.—L. K., a woman aged 35, had vision of 20/50 in the right eye with a correction of —0.50 D. sph. ⌒ —2.75 D. cyl., axis 180, and of 20/200 in the left eye with correction of —0.50 D. sph. ⌒ —3.00 D. cyl., axis 175. The fields were uniformly contracted. The patient had had night blindness since early childhood.

Examination of the fundus revealed retinitis pigmentosa in both eyes; the appearance of the right eye differed little from that of the left. There appeared all the characteristics of progressive pigmentary degeneration of the retina: At the periphery, patches of irregularly shaped black pigment were observed, together with atrophic discoloration of the optic nerve, and thinning of the retinal vessels to such a degree that they were barely visible.

Contact lenses were prescribed, and the patient was observed during one year. Vision was improved to 20/40 in the right eye and to 20/70 in the left eye. With these lenses she was able to see many more details, although repeated tests failed to demonstrate any extension of the limited field of vision. At first, the resulting correction was insufficient to overcome the disinclination of this nervous patient to

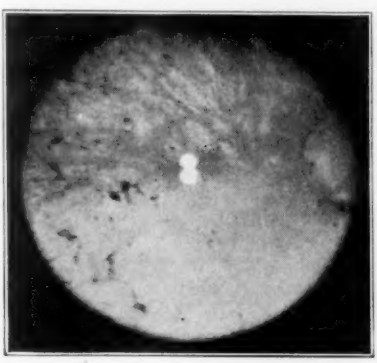

Fig. 5 (case 4).—Photograph of the right fundus, showing retinitis pigmentosa.

wear contact glasses for extended periods. She is, however, slowly improving in this respect, and is receiving increasingly greater benefit from her contact lenses.

CONCLUSIONS

Even though contact lenses cannot remedy existing pathologic changes in the fundus, their use in cases in which this condition complicates myopia has led to promising results. Improvement of vision was obtained in each case of the series reported here.

From experience in the present cases, success would seem to depend on two definite prerequisites—the one objective and the other psychologic. First, contact lenses, in order to be tolerated by the patient, must produce considerably greater correction of visual disturbances than spectacles; second, the patient must be willing to undergo a period of trial and accommodation to the new glasses. Only if these two conditions are fulfilled will the prescription of contact lenses prove to be entirely satisfactory.

SUMMARY

Contact lenses were prescribed in 30 cases of myopia concomitant with degenerative conditions of the fundus of the eye. The data on 12 cases, presenting more pronounced pathologic changes are presented, 4 of which are described in detail.

In each case, vision was greatly improved as to clarity and detail of objects perceived.

Contact glasses were well tolerated by all patients; however, in most cases the ease with which they were tolerated was directly related to the benefit which they afforded.

The reasons for the improvement of vision through the use of contact lenses in selected cases of myopia are discussed.

In selected cases, contact lenses are superior to spectacles in the correction of deficient vision due to myopia concomitant with a pathologic condition of the fundus.

Correspondence

RECENT ADVANCES IN THE PHYSIOLOGY OF VISION

To the Editor:—In an article entitled "Recent Advances in the Physiology of Vision—Part II," published in the *British Medical Journal* (**1:** 913-916 [June 28] 1947), the author, H. Hartridge, protests against the continued acceptance of the so-called trichromatic theory of color vision, first postulated by Thomas Young. The value of his report to the ophthalmologist, however, will probably lie in the information concerning recent researches on color vision rather than in the arguments for or against any one type of color theory. Hartridge believes that the results of more critical methods of research demand that the three receptor form of theory be abandoned in favor of some polychromatic form. Special reference is made to Granit's work, reviewed by the author in Part I of this report (*Brit. M. J.* **1:** 637-639 [April 27] 1946), in which a microelectrode technic was applied to individual nerve fibers of the retina of many mammals to determine spectral response curves. Granit observed as many as seven response curves in the frog and four in the cat. The seven "modulators" observed in the frog fall into three main groups, corresponding with red, green and blue light. Hartridge believes this grouping may explain in part why vision has so long been thought to be trichromatic.

Stimulated by Granit's work, Hartridge attempted a microstimulation of the human retina by utilizing "tenuous beams of light" sufficiently narrow to subtend at the eye an angle of only 8 seconds of arc and to form on the retina, he claims, geometric images equal to one-fifth cone unit. (One "cone unit," according to Hartridge, subtends an angle at the observer's eye of 41.236 seconds of arc.) Employing a finely adjustable fixation light, the geometric image of which equaled less than one-tenth cone unit, he found that the fixation point for maximal response to different spectral rays was not the same. In all, seven different fixation points were located, namely, those for red, orange, yellow, green, blue-green, greenish blue and blue. As determined on three occasions, the mean values of these locations varied by less than one-fourth cone unit. However, a beam of white light remained white in appearance wherever it was situated on the retina. This work, as well as Granit's, suggests that there are at least seven individual receptors. To be fully understood and appreciated, the details of the retinal microstimulation technic must be read in Hartridge's longer report (*Phil. Tr. Roy. Soc., London, s.B* **232:** 519-671 [May] 1947). Questions will arise in the reader's mind as to the possibility of such a minute control of fixation, especially in the light of the unsteadiness of the eye's fixation found by Adler and Fliegelman (Influence of Fixation on Visual Acuity, ARCH. OPHTH. **12:** 475-483 [Oct.] 1934). Results are given only for Hartridge's left eye. I have not been able to find any statement that other observers were used.

Hartridge amplifies his thesis with arguments drawn from other fields of color response. In section II he discusses color mixture experiments and contends that the reduction of saturation undergone by mixed colors as compared with the matching monochromatic color and the resultant need to employ negative values to procure a perfect match suggest that color vision is polychromatic (as opposed to trichromatic). As he develops the situation, components of seven spectral colors are required to match perfectly the extreme red, the yellow, the blue-green and the extreme violet of the spectrum, taken as examples. In section III he considers briefly the various types of color blindness, all of which, he believes, cannot be explained by the partial or complete deficiency of a single sensation mediated by one of three receptors. In this connection, he does not discuss Wright's alternative suggestions for explaining these situations on a trichromatic basis (Wright, W. D.: Researches on Normal and Defective Colour Vision, London, Henry Kimpton, 1946, pp. 352-354). Section IV deals with the inconstancy of the receptor curves determined with foveal and extrafoveal stimuli and with subjects having certain color deficiencies, and section V, with data on hue discrimination. It is unfortunate that in this section he illustrates his arguments with a hue discrimination curve for "one subject with normal colour vision," since this curve departs markedly from the careful determinations of other investigators, including the recent work of Wright and Pitt. To explain the troughs and peaks found in this hue discrimination curve, Hartridge concludes that color vision must be mediated by not less than five types of receptor. This conclusion is at variance with that of Wright (Researches on Normal and Defective Colour Vision, London, Henry Kimpton, 1946, p. 172 and Pitt (*Proc. Roy. Soc., London, s.B* **132:** 113-115, 1944), who find the trichromatic theory adequate to explain the shape of the hue discrimination curve. Sections VI and VII are devoted to the difficulty in explaining, on a trichromatic basis, the changes in hue undergone by colors (*a*) by reduction of visual angle, (*b*) by reduction of illumination and (*c*) by the use of peripheral vision. These changes, he believes, could be explained by assuming that foveal vision is mediated by at least four receptors: red, orange, blue-green and greenish blue—the addition of yellow and blue receptors constituting full color vision in the parafoveal region—and, in order to explain the invariable appearance of white as white, by assuming that these receptors are present in complementary pairs. The dropping out of complementary receptors in pairs would, then, account for the changes in hue observed. Since, however, the changes in hue experienced under the conditions noted by Hartridge are markedly dependent on the brightness ratio between color and its surround, the argument here is not entirely convincing. This is the case also in a later correspondence item by Hartridge (*Brit. M. J.* **2:** 69 [July 12] 1947), in which he states that if small test objects of bright red, bright green and lemon yellow are placed on a black background and viewed at a distance, the yellow is seen as pale gray or white, while the red and green retain their color. This phenomenon, he claims, argues against the trichromatic theory, which explains yellow as a mixture of red and green. Experiments of this type also, however, yield results which

depend on the brightness ratio between color and background, as well as on the chroma of the colors compared. When, for example, Munsell pigments of equal chroma and value are placed on a gray surround of the same value and viewed at a distance, the order of disappearance of red, yellow and green is by no means the order found by Hartridge.

The physiology of vision is a difficult and controversial subject. Hartridge's article is challenging and will serve to arouse controversy. This, no doubt, was part of his intention.

GERTRUDE RAND, PH.D., New York.

Institute of Ophthalmology, College of Physicians and Surgeons.

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

National Study of Congenital Malformations and Maternal Infection.—In an effort to collect more precise data on the relations between certain maternal infections and congenital malformations, a nationwide study is being sponsored by the American Academy of Pediatrics and the National Society for the Prevention of Blindness, Inc. Questionnaires are being sent to obstetricians, ophthalmologists and pediatricians, seeking the reporting of cases of rubella in expectant mothers and of children with congenital defects that might be attributed to other infections in the expectant mother, such as measles, chickenpox, mumps and influenza.

Although an association has been established between the occurrence of rubella early in pregnancy and certain congenital defects in the offspring, information is lacking as to the frequency with which this happens and as to the possible influence of other communicable diseases that might have been contracted by the expectant mother.

Data will be studied by the following committee: Herbert C. Miller, M.D., Kansas City, Kan.; Stewart Clifford, M.D., and Clement A. Smith, M.D., both of Boston; Josef Warkany, M.D., of Cincinnati; James Wilson, M.D., of Ann Arbor, Mich., and Herman Yannet, M.D., of Southbury, Conn. Physicians knowing of cases are urged to register them with Dr. Miller, chairman of the committee.

SOCIETY NEWS

Ohio State Optometric Association.—On Wednesday afternoon and evening, November 5, the Dayton Safety Council of the Chamber of Commerce and zone 9, Ohio State Optometric Association, are cosponsoring a familiarization "Seminar on Industrial and Occupational Vision," to be held at the Van Cleve Hotel, Dayton, Ohio. All members of the ophthalmologic professions are cordially invited to attend. The seminar will start at 2:30 p. m., with the program as follows:

2:30 p. m.: Introduction: Occupational Vision, Dr. F. E. Billette, president, zone 9; Dr. R. N. Roth, seminar chairman

3:00 p. m.: Proneness to Accidents and the Employee's Vision, N. C. Kephant, Ph.D., La Fayette, Ind.

3:30 p. m.: Industry and Seeing—One Aspect of Occupational Seeing, Joseph H. Tiffin, Ph.D., La Fayette, Ind.

4:00 p. m.: The Nurse in the Industrial Vision Program, Mrs. Hazel H. Leedke, R.N., Kaukauna, Wis.

4:30 p. m: A Testing and Training Program Utilizing Visual Classification, Correction and Placement of Employees, Mr. Dave Ekberg, Dayton, Ohio

5:00 p.m.: Aspects of a Plant Safety Goggle Program, Mr. Bill Carroll, Columbus, Ohio

7:00 p.m.: Dinner, to be followed by round table discussion and question and answer period.

The registration fee is $5 per person, including cost of dinner. Reservations may be made with Dr. R. N. Roth, 2324 Salem Avenue, Dayton, Ohio.

German Ophthalmological Society of Heidelberg.—The German Ophthalmological Society of Heidelberg is making preparations for resuming its meetings in Heidelberg in 1948, the last meeting having taken place in Dresden in 1940.

The society has published in the past seven years the following three books: von Tschermak-Seysenegg, A.: Einführung in die physiologische Optik, 1942; Velhagen, K.: Sehorgan und innere Sekretion, 1943, and Lauber, H.: Das Gesichtsfeld, 1944.

Further information can be obtained from Dr. E. Engelking, Universitäts-Augenklinik, Heidelberg, Germany.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

General Diseases

THE SYNDROME KNOWN AS "REITER'S DISEASE" (A TRIAD OF POLYARTHRITIS, URETHRITIS, AND CONJUNCTIVITIS). W. P. U. JACKSON, Brit. M. J. **2:** 197 (Aug. 10) 1946.

The patient is usually a young adult with an irregular fever and one or more of the three symptoms indicated. The conjunctivitis is bilateral and catarrhal or profusely purulent, with episcleritis and sometimes iritis or keratitis. No organism can be noted in conjunctival smears. The arthritis affects the large joints, with pain, and is often flitting. Synovitis is frequent; the fluid may be purulent but is always sterile. The urethral discharge is abundant and purulent, without any organism, and is sterile on culture. The disease sometimes begins with diarrhea and a generalized rash. Complement fixation tests for gonorrhea and Wassermann tests give negative reactions. The disease is self limited, though subject to recurrence. This syndrome is important, as it resembles a gonococcic infection, but with the latter there is a history of contact and of urethritis which precedes the other symptoms. The gonococcus is present except in the conjunctival discharge. The fixation test usually gives a positive reaction, and the responses to treatment in the two conditions are different. No treatment seems to be of particular avail for Reiter's disease except to put the patients to bed for six weeks to three months. Three cases are reported.

ARNOLD KNAPP.

Glaucoma

PERSONALITY PATTERNS OF WHITE ADULTS WITH PRIMARY GLAUCOMA. H. L. HIBBELER, Am. J. Ophth. **30:** 181 (Feb.) 1947.

Hibbeler, in a preliminary survey of patients wtih primary glaucoma, found that males tended to depression and hysteria and females to paranoia and schizophrenia.

W. S. REESE.

GONIOTOMY FOR CONGENITAL GLAUCOMA: URGENT NEED FOR EARLY DIAGNOSIS AND OPERATION. O. BARKAN, J. A. M. A. **133:** 526 (Feb. 22) 1947.

Barkan makes a plea for the early recognition of congenital glaucoma, since if goniotomy is performed soon after discovery of the condition the symptoms can be relieved and the pressure in most cases permanently normalized, with resulting useful vision and normal appearance. Of 76 eyes with infantile glaucoma on which goniotomy was performed, pressure was normalized in 66, with vision maintained or restored over periods of from one to ten years. In 10 eyes the operation was unsuccessful. Glaucomatous atrophy of the optic nerve was the exception in this series. It occurred only in the few cases in which normalization of pressure was unduly delayed. In 1 case the excavation

of the nerve head almost disappeared after normalization of pressure. The abstracter has reported the occurrence of this unusual sequel to sclerocorneal trephining in a case of chronic simple glaucoma.

J. A. M. A. (W. ZENTMAYER).

Injuries

A CASE OF A SPLINTER OF GLASS IN THE ANTERIOR CHAMBER OF FOUR YEARS' DURATION. W. B. DOHERTY, Am. J. Ophth. **30: 177** (Feb.) 1947.

Doherty reports a case in which a splinter of glass in the angle of the anterior chamber apparently disappeared after operation. He remarks the difficulty encountered in such cases and suggests the use of reflexless instruments. He has had several of these made.

W. S. REESE.

Lens

EXTRACTION OF DISLOCATED LENSES BY THE HIGH FREQUENCY NEEDLE OF JESS AND LACARRÈRE. A. GARDILČIĆ, Ophthalmologica **112:255** (Oct.-Nov.) 1946.

Jess recommended extraction of lenses which are dislocated into the vitreous by means of a high frequency electric needle. The present report is a modification of Jess's method. A conjunctival flap is thrown down, and before the section is made a high frequency needle is inserted through the limbus into the interior of the eye. It is then pushed into the lens and the current is switched on. This is done under control with an ophthalmoscope or with transillumination. The needle becomes adherent to the lens by virtue of coagulation of the lens protein. After coagulation occurs, the incision is made for the extraction and the lens or cataract carefully removed. Corneoscleral sutures, which were placed at the beginning of the operation, are then tied, and the conjunctival flap is sutured in place. One case is reported. F. H. ADLER.

Methods of Examination

OBJECTIVE TESTING OF RETINAL FUNCTION BY THE ELECTRORETINOGRAM IN A CASE OF CONCENTRIC CONTRACTION OF THE VISUAL FIELD. M. MONNIER and M. AMSLER, Ophthalmologica **110: 225** (Nov.-Dec.) 1945.

Electroretinograms have been obtained on human beings by a number of investigators. Monnier and Amsler describe their technic, which they applied in a clinical case, that of a woman aged 52, who had had an accident without direct injury to the skull, and who showed as a result of this trauma concentric narrowing of the visual fields, diminution of central visual acuity, insufficiency of convergence, fatigability during reading and muscae volitantes.

The visual fields changed on various occasions, but never measured more than 60 degrees temporally and 35 degrees nasally. Both pupils

reacted to light, and adaptation of the retina was normal. The electroretinogram for this patient, taken from the peripheral portions of the retina, which were apparently blind, showed normal excitability, so that the authors believe that the impulses were normally discharged from the retina into the optic nerve. They conclude that these discharges did not reach the visual centers but were stopped at either the lateral geniculate bodies or the level of the synapses in the occipital cortex.

F. H. ADLER.

Neurology

PSYCHOLOGICAL REACTIONS IN WAR-BLINDED. W. M. HARROWES, Brit. M. J. 2: 129 (July 27) 1946.

This is a brief review of the cases of 40 war-blinded men who, while undergoing training for reconstitutive processes, such as Braille reading, typing, shorthand, woodwork and basketmaking, were studied in an attempt to discover the indications presented by each man for types of future occupation. In addition, psychiatric examinations were carried out along the lines of Adolf Meyer's personality study. Three late reactions to loss of vision were noted: (*a*) tension, (*b*) overcompensation and (*c*) restabilization.

ARNOLD KNAPP.

AMBLYOPIA CUM POLYNEUROPATHIA CAUSED BY STARVATION. A. W. MULOCK HOUWER, Ophthalmologica 112: 177 (Oct.-Nov.) 1946.

The author, who was formerly the head of the department of ophthalmology at the University of Batavia, Netherlands East Indies, was interned by the Japanese from March 1942 until September 1945. During his internment in the various prison camps, he had an opportunity to observe a number of patients who exhibited a syndrome which he calls "camp eye." The patients complained of difficulty in reading and of inability to recognize persons at a rather short distance. Examination showed a reduction of central visual acuity and, in every case, a central or paracentral scotoma.

In the early cases the scotoma was relative; in the very serious cases it was absolute. The fundi were normal save for slight changes in the macular area, which consisted of absence of the foveolar reflex, even in patients under 40 years of age, a slight granular appearance of the foveola and drusen of Bruch's membrane. The patients had nearly all experienced abnormal sensations in the feet, legs, hands or forearms for some time. These sensations consisted of a prickly feeling or of an abnormal sensation of heat or cold. A few patients had nutritional edema. In some patients hemorrhages were present in the inner layers of the retina. In some of the more advanced cases, pallor of the temporal part of the optic disk developed. The author believes that the condition in these cases of "camp eye" is analogous to the toxic amblyopias and the amblyopia in serious cases of hyperemesis gravidarum.

He considers that the chief cause of the disease is a lack of vitamin thiamine. A deficiency in other vitamins of the B complex and a

generally poor diet, consisting mostly of carbohydrates, are favorable to the development of the syndrome. The patients with recent onset of the disease recovered under treatment with large doses of thiamine hydrochloride. The author advises the administration not only of thiamine hydrochloride but also of the other vitamins of the B complex, especially nicotinic acid. The diet should be poor in carbohydrates, as the disproportion between the high intake of carbohydrates and the deficiency of vitamin B may play an essential part in causing the central scotoma. Sodium nitrite may be given for vasodilatation to speed up recovery.

F. H. ADLER.

Ocular Muscles

PATHOGENESIS AND TREATMENT OF CONCOMITANT STRABISMUS. A. DEL BARRIO, Arch. Soc. oftal.hispano-am. **6**: 863 (Sept.) 1946.

The author expresses the belief that strabismus is caused by the absence or insufficiency of the retinal convergence reflex, which is developed in the first years of life. At that age there is great power of accommodation, which produces an excess of convergence; but if the retinal convergence reflex is strong enough it can maintain the normal position of the eye and binocular vision. However, if the reflex is weak, binocular vision is lost and convergent strabismus appears.

In cases of divergent strabismus there is an insufficient retinal convergence reflex, which manifests itself after the age of 8 or 10 years, being compensated for before that time by the excessive accommodative power, which acts on convergence. Only when accommodation begins to weaken after that age is binocular vision lost and the eye which is in divergence seeks the position of rest. Errors of refraction act as an acessory factor, as well as everything which diminishes vision or in any way alters the physiologic sensorial or motor relations.

Optical treatment rarely modifies the degree of squint; it may, however, improve vision when associated with temporary occlusion of the fixing eye. After the age of 12 years the treatment of squint should be surgical and functional.

Surgical treatment must produce an overcorrection of 10 or 15 degrees in patients with convergent squint up to 20 years of age and of 5 degrees after that. With divergent squint a slight convergent position should be the endeavor.

Functional treatment is indicated only in cases in which the vision of the amblyopic eye is above 1/10. At the beginning the orthoptic training should be given without any instrument; a light and a colored glass are sufficient. The author gives in detail his method of functional treatment and claims that in a few days, with patients of fair intelligence, binocular vision is gained. Later, exercises with the stereoscope and the diploscope are given, and, finally, Javal's exercises for controlled vision are performed. The average duration of the course of functional treatments is one or two weeks, daily sessions of a half-hour being given in the office and the patient repeating the exercises at home.

H. F. CARRASQUILLO.

Operations

ARTIFICIAL THICKENING OF THE CONJUNCTIVAL FLAP IN FISTULIZING OPERATIONS. A. P. FILHO and S. R. SEBAS, Brasil med. **59:** 1 and 7 April 1945.

The relative frequency of intraocular infection following fistulizing operations is mentioned. Sections of sheep eye showed thinning of the conjunctival covering of the fistula. To increase the thickness of the flap, the submucous tissue is utilized by bringing it over the fistula.

M. E. ALVARO.

Orbit, Eyeball and Accessory Sinuses

PULSATING TRAUMATIC ENOPHTHALMOS: REPORT OF A CASE. S. KALFA, Vestnik Oftal. **25:** 41, 1946.

A patient aged 24 had been injured four years previously in the left orbit. Anophthalmos and ptosis followed the injury. An unsuccessful attempt was made in another hospital to correct the ptosis. There was a defect in the upper outer wall of the orbit. The eye presented enophthalmia (5 mm. with Hertel's exophthalmometer), marked esotropia, limitation of movements outward, ptosis and pulsation of the eyeball, visible through the eyelids. No bruit was elicited. A tenotomy of the left medial rectus muscle straightened the eyeball. After this tenotomies were performed of the other rectus muscles, but the enophthalmos remained unchanged.

Kalfa believes that the enophthalmos in this case was due to dislocation of the fragments of the upper orbital wall with displacement of the orbital fat into the skull. The pulsation of the eyeball was caused by the defect in the upper orbital wall, through which the pulsation of the brain was transmitted to the eyeball. The absence of bruit and of dilated veins in the eyeball confirms this view. This condition could be considered as spurious exophthalmos.

O. SITCHEVSKA.

Parasites

SUBCONJUNCTIVAL CYSTICEROSIS. G. BURSUK, Vestnik oftal. **25:** 40, 1946.

A woman aged 33 showed on examination a round, firm swelling at the upper temporal region of the right eyeball under the conjunctiva, which seemed to be attached to the palpebral part of the lacrimal gland. The eyeball was normal otherwise; vision was 1.0. All tests, including a roentgenographic examination, gave normal results except for an increase of eosinophils in the blood (9 per cent). The patient was scheduled for operation, but on that day she presented a semitransparent, milky white vesicle, which came out of the eyeball. On examination the head of the worm was found; thus the cysticercus perforated the conjunctiva. Cysticercosis of the eyeball is rare. In Russia 117 cases of this condition were recorded in sixty-five years. In Leningrad only 1 case was found among 70,000 patients with ocular disease. This case is reported because of the unusual manner of self expulsion through the conjunctiva.

O. SITCHEVSKA.

Pharmacology

SIGNIFICANCE OF ACTION OF PAREDRINE ON THE OCULAR TENSION OF RABBITS. E. SCHMERL, Am. J. Ophth. **30:** 187 (Feb.) 1947.

Schmerl found that paredrine hydrobromide aqueous (a 1 per cent solution of *p*-hydroxy-α-methylphenylethylamine hydrobromide in distilled water, made tear isotomic with 2 per cent boric acid and preserved with merthiolate, 1:50,000) applied locally to the pericorneal vessels of rabbits caused increased intraocular tension, dependent on the vascular bed and the quantity of pericorneal vessels constricted.

W. S. REESE.

The Pupil

MIOSIS CONGENITA. J. C. HOEST, Acta ophth. **20:**293, 1942.

The author discusses the previous reports of Hoeth and Berner on miosis congenita. One of Hoeth's patients was reexamined after an interval of twenty years. The pupils remained very small and were without reaction. The patient had poor vision, day blindness and spasm of accommodation, which was helped by the regular use of atropine.

In a case of Redslob's, with histolologic study, the musculature of the iris showed defective development. In another case of the author's there were absence of the dilator muscle and an atypical ciliary muscle, much of the muscle being found in the periphery of the iris.

O. P. PERKINS.

Physiology

PERMEABILITY OF THE BLOOD-AQUEOUS BARRIER OF THE NORMAL EYE TO FLUORESCEIN. WALTER HAEFELI, Ophthalmologica **112:** 226 (Oct.-Nov.) 1946.

A method was devised by Amsler and Huber (*Ophthalmologica* 111: 156, 1946) by means of which the amount of fluorescein could be estimated in the anterior chamber of the intact eye. These authors found, contrary to the previous opinion of investigators, that fluorescein, even in the normal eye, appeared in the anterior chamber in measurable quantities. Although the amount varies somewhat from one subject to another, the normal limits have been well established, which Haefeli speaks of as "the normal band."

The present paper deals with the further determination of this normal band. Two cubic centimeters of a 10 per cent solution of fluorescein sodium is injected into the antecubital vein. The time of the injection is recorded, and the time of the appearance of the fluorescein in the anterior chamber is determined with a slit lamp beam and observation through the binocular microscope. By introduction of a resistance, the intensity of the light is reduced to the point at which no green coloration can be seen in the aqueous. A direct proportionality exists between the concentration of fluorescein in the anterior chamber and the strength of current, in amperes, of the light

source at which the fluorescein just becomes visible. Curves are constructed in which the abscissas represent the time and the ordinates the values, in amperes. Two hundred graphs of the normal band are given. The author finds that fluorescein passes into the aqueous humor approximately four minutes after intravenous injection. In children under 15 years the fluorescein appears more quickly. In general, the maximum concentration of fluorescein is reached in twenty-five to thirty minutes. In any one subject, the two eyes show the same permeability. Fluorescein remains in the anterior chamber for several hours, considerably longer than in the blood stream. The permeability determined at different times varies only slightly for the same person. When injected in smaller quantities than usual, fluorescein passes more slowly into the aqueous but attains almost the same concentration. The fluorescein enters the anterior chamber free of albumin.

F. H. ADLER.

Refraction and Accommodation

MYOPIA DUE TO A SULFONAMIDE DRUG: REPORT OF A CASE, WITH EXPLANATION OF ITS PATHOGENESIS. O. VON FIEANDT, Acta Ophth. **20:** 24, 1942.

A careful study was made of a case in which myopia developed after ingestion of a sulfonamide drug. By atropinizing one eye only, determining the refraction and the accommodative power of the two eyes and observing the subsequent behavior after instillation of atropine and physostigmine, the author reaches the conclusion that the myopia is the result in part of a spasm of the ciliary muscle and in part of changes in the lens of the eye.

O. P. PERKINS.

Retina and Optic Nerve

THE PERIOPTIC ATROPHIC RING AND ITS RELATIONSHIPS. R. PICKARD, Brit. J. Ophth. **30:** 437 (Aug.) 1946.

The cause of the atrophic rings which are occasionally observed around the optic disk was studied in 826 consecutive patients over 50 years of age. The eyes were classified according to the presence or absence of atrophic rings and the normality or narrowing of the retinal vessels. If the narrowing of the vessels was present in any part, that eye was classified as having narrow vessels. The only lesions common to cavernous atrophy, glaucoma and atheroma were the narrowed vessels and the rings. The theory is proposed that the rings and the cavernous atrophy are the consequence of a change in the circle of Zinn, analogous to that which occurs in the retinal vessels. On the other hand, the changes in glaucoma are due to pressure and consequent narrowing of the vessels or pressure on the nerves as they pass around the cup. If the cavernous atrophy is due to vascular trouble, operation will do no good, and this was true in a few cases in which it was tried.

W. ZENTMAYER.

THROMBOSIS OF THE RETINAL VEIN TREATED WITH HEPARIN: REPORT OF 30 CASES. ROSENGREN AND SFENSTROM, Acta ophth. **20:145**, 1942.

In 30 cases thrombosis of the retinal vein was treated with injections of heparin daily for an average period of ten days. In 10 cases the thrombosis was in the trunk and in 20 cases, in branches of the vein. Improvement was obtained in 21 cases; the condition became worse in 5 cases and remained unchanged in 4 cases. The thromboses of the trunk were comparatively little influenced. The average improvement in visual acuity was from 0.239 to 0.375. This slight improvement was less evident two to four weeks after cessation of the treatment.

O. P. PERKINS.

Trachoma

THE TREATMENT OF TRACHOMA. E. O. MARKS, Brit. J. Ophth. **30:** 213 (April) 1946.

For the past twenty-five years Marks has had the care of trachomatous children at the Hospital for Sick Children and at the Wilson School Ophthalmic Hostel, at Brisbane, Australia. The children came from the dry inland areas of Western Queensland, where trachoma is prevalent. The differential diagnosis was rendered somewhat difficult because in those areas the dryness of the atmosphere, the dust and the glare combine to form irritating conditions for the conjunctiva, with consequent common irritative conjunctivitis. In running tests in which the two, seemingly equally affected, eyes were treated by different local methods, it did not appear to matter what one applied locally, other than lavage to keep the eyes clean, with perhaps the exception of the sulfonamide drugs. These substances, when administered orally, were of great value in treating secondary infections but did not benefit the trachoma. They seemed less efficient in local application.

W. ZENTMAYER.

TREATMENT OF TRACHOMA WITH STREPTOCIDE [SULFANILAMIDE]. A. VOLOKITENKO, Vestnik oftal. **23: 32**, 1944.

The mechanism of the action of sulfonamide drugs in treatment of trachoma can be explained by the activation of the reticuloendothelial system. Experimental work confirmed this view, as Volokitenko obtained the growth of young fibroblasts on sections of the third lid in rabbits after injecting into them a diluted cytotoxic-antireticuloendothelium serum.

Thirty patients with various stages of trachoma were treated with gradually decreasing doses of sulfanilamide. In the majority of patients the blood picture showed a decrease of leukocytes and of hemoglobin. The author concludes:

1. Complete cure was obtained in 60 per cent of patients with trachoma who were treated with sulfanilamide. 2. The daily dose of sulfanilamide should not exceed 1.0 to 1.5 Gm. 3. In some cases complete cure was obtained in only two to three months. 4. Vision was improved in all but 4 patients. 5. The local application of sulfanilamide drops had no bearing on the therapeutic results, so that the drug was

given only internally. 6. The best results were obtained in the granulation stage of trachoma. Flattening of the granules was observed on the eighth day and their absorption in two to three weeks. The pannus began to disappear about the fifteenth day. O. SITCHEVSKA.

Tumors

MELANOMAS OF THE CHOROID. C. d'AQUILA DE CASTAÑE DECOUD, An. argent. de oftal. **5:** 123 (Oct.-Dec.) 1944.

Seventeen cases of melanoma occurring in patients operated on during a period of ten years at the department of ophthalmology of the Faculty of Medicine of Rosario are described pathologically by the chief of the laboratory. A classification is made on the basis of cellular elements and the pigment content.

The following summary is given: 1. Of a total of 33,694 cases, there were 17 cases of melanoma of the choroid, a proportion of 0.05 per cent. Of these tumors, 15 were pigmented and 2 were achromic. Of tumors composed of cells of the same type, those with pigment were notoriously more malignant. 2. The range of ages was from 40 to 80, the tumor being more frequent between the ages of 40 and 60. 3. In 7 cases the tumor was located at the equator of the eyeball; in 6, in the anterior segment; in 2, in the posterior segment, and in 2 cases the position was not mentioned. 4. The patients were followed up to the time of the report, the number of years varying from two to ten. 5. The tumor is usually round and circunscribed and surrounded by melanophores of the choroid, which form a membrane, the tumor herniating through this membrane in some cases. 6. According to the type of predominant cell, the tumors were classified as follows: round cell, 8 cases; fusiform cell, 6 cases; mixed cell, 2 cases, and unclassified, 1 case. Extraocular extension of the tumor was noted in 4 cases. The article is illustrated with photomicrographs. H. F. CARRASQUILLO.

Uvea

MENOPAUSAL IRIDOCYCLITIS. M. E. REDSLOB, Ann. d'ocul. **178:** 225 (June) 1945.

The etiology of iridocyclitis is discussed. In about 25 per cent of cases the etiologic factor cannot be determined. Within the past few years some progress has been made. With improvement in diagnostic facilities, various etiologic agents have been uncovered. The author is particularly interested in the susceptibility of women to iridocyclitis, especially during the menopause. The physiology of the estrogens is discussed. When the normal endocrine balance is disturbed, as in the menopause, the female is more prone to infections. There is some similarity to diabetes, which is an endocrine disturbance. It is well accepted that diabetic persons are more susceptible to infections than nondiabetic persons. A truly diabetic iritis is rarely seen, though iritis due to other causes is frequently seen with diabetes.

The author mentions the work of Dejean, who expressed the belief that in the majority of cases iridocyclitis is due to tuberculosis. This

conclusion is based on response to therapy rather than on clinical proof. Redslob, however, feels that the iritis occurring during the menopause is probably of syphilitic origin. The syphilis may be masked but, nevertheless, is probably the cause.

Two cases of iridocyclitis in women aged 49 and 50, respectively, are reported. Both patients were having menopausal symptoms. The first had congenital syphilis and the only therapy that helped the second was neoarsphenamine.

P. R. McDONALD.

THROMBOPHLEBITIS AS A FOURTH SYMPTOM OF RECURRENT IRITIS WITH HYPOPYON. B. ADAMANTIADES, Ann. d'ocul. **179:** 143 (March) 1946.

Ophthalmologists and dermatologists have for some time been interested in the syndrome of recurrent iritis and hypopyon. Previous to this report the cases reported have had in common iritis, aphthous ulcers of the mouth and genital ulcers. The author has recently seen 2 cases in which retinal hemorrhages of the type associated with thrombophlebitis were present. In reviewing the previously published cases, he found that this lesion had been frequently noted but no particular attention had been paid to it.

The author reports 2 cases that presented the lesions characteristic of this syndrome. The etiologic basis of the condition has never been settled. Some consider it to be tuberculous; others, a staphylococcic infection. Because of the periodicity of the iritis, it is felt that the disturbance must have an allergic basis.

The disease resembles periodic ophthalmia of horses and may be caused by a filtrable virus or by brucellosis.

P. R. McDONALD.

PULMONARY CHANGES IN CHRONIC AND ACUTE IRIDOCYCLITIS, SCLEROKERATITIS AND CHOROIDITIS. L. ESSEN-MÖLLER, Acta ophth. **20:** 97, 1942.

This article deals statistically with roentgenologic findings in a control group of 442 cases and in 186 cases of chronic iridocyclitis, 140 cases of acute iridocyclitis, 62 cases of sclerokeratitis and 44 cases of choroiditis.

In cases of chronic iridocyclitis, active changes in the hilus were found oftener than in the control group. Active, or even healed, changes in the lungs proper were not definitely more frequent. Nor were any changes noted more frequently in the cases of acute iridocyclitis. The cases of sclerokeratitis showed an increased incidence of healed pulmonary lesions. The choroiditis group did not show an increased incidence of any type of pulmonary lesion.

O. P. PERKINS.

PROGNOSIS OF CHRONIC IRIDOCYCLITIS. L. ESSEN-MÖLLER, Acta ophth. **20:** 121, 1942.

The author made a statistical survey of 244 cases of chronic iridocyclitis treated at the eye clinic of Lund University between 1928 and 1939. In 1938, reexamination in 211 of these cases showed healing in 70 per cent and actitve iridocyclitis in 30 per cent. The tendency to

healing was greater in cases of unilateral than of bilateral involvement and was perhaps a little better in males than in females. However, in only 45 per cent of cases did the eyes heal without residual complications which reduced vision; in 42 per cent visual acuity was less than 0.3; in 30 per cent the patient became practically blind, and a diminished earning capacity of 80 per cent to 100 per cent was the result in 20 per cent.

The visual acuity and the person's earning capacity tended to become less the longer the period of observation.

Increased intraocular tension was observed in one third of the cases. If this was slight and transitory, it did not affect the prognosis, but if it were high or prolonged, the prognosis was less good, particularly if operative intervention was necessary.

O. P. PERKINS.

Vision

PSYCHOLOGICAL REACTIONS IN SOLDIERS TO THE LOSS OF VISION OF ONE EYE, AND THEIR TREATMENT. P. M. DUKE-ELDER and E. WITTKOWER, Brit. M. J. **1**: 155 (Feb. 2) 1946.

The authors begin with the statement that the loss of one eye is not in itself a severe disability; at the same time, persons who have lost an eye are often unduly distressed about their defect and feel handicapped in numerous activities out of proportion to their actual disablement. Their handicaps are not anatomic in origin but are due to what is commonly described as a functional overlay. The war has developed conditions in which study of the reactions of persons who have lost one eye is made possible; these conditions are important, as the same reactions are met in civilian life.

A series of 102 patients were examined: The disability in 39 was due to disease and in 63 to injury. Twenty-six of these men were seen within six months of the onset of the disease or the occurrence of the injury; 15 had chronic ocular disease; 61 had been blind in one eye as the result of disease or of injury for at least six months.

The emotional reaction to monocular injury and disease is then described. The emotional reaction to permanent blindness in one eye was studied. In 47 of 61 men there were evidences of morbid fears concerning their social relationship, the future economic security of their dependents and the possibility of further harm to themselves. These reactions were classified as follows: social anxiety, 26 men; anxiety over dependents, 10 men; self-centered and self-pitying anxiety, 11 men.

The factors which determine the emotional reactions are (1) previous personality, (2) nature of the disability and (3) nature of employment.

The authors conclude that the psychologic effect of the loss of an eye is out of proportion to the actual physical disability. Of this group, 78 per cent showed abnormal psychologic reactions. The fear of blindness is so general that the loss of one eye causes undue anxiety as to the remaining eye. This leads to certain symptoms referable to the good eye, such as eyeache, eyestrain, photophobia and night blindness. These functional symptoms, rather than the actual defect,

are responsible for the impairment in efficiency of the one-eyed man. Adequate treatment of these patients is not completed with the medical attention to the eye. The psychologic treatment should be begun and carried on not only in the hospital but also in convalescence. During the latter period Duke-Elder and Davenport (*Brit. J. Phys. & M. Indust. Hyg.*, **5**: 88, 1942) stated that "rapid restoration of physical fitness, training to compensate for the loss of binocular vision and to readjust the faculty of orientation, and measures to overcome the psychological distress of mutilation are essential."

Finally, attention is drawn to the importance of improving the occupational prospects of the one-eyed worker by means of impressing on the public and on private employers the negligible loss of efficiency in most vocations in the case of the one-eyed worker.

This is a very well written and important article, which merits careful reading and consideration.

ARNOLD KNAPP.

Therapeutics

PENICILLIN IN CAVERNOUS SINUS THROMBOSIS. A. G. VASIUTINSKY, J. Ophth. **1**: 25, 1946.

An officer of the Soviet army, aged 24, had severe thrombosis of the right and left cavernous sinuses, the cause being a furuncle of the nose. There were rigidity of the neck and hemiparesis. Vision was limited to light perception in each eye.

Penicillin was injected (15,000 units every four hours) for a period of four days. The temperature was normal in eight days; the exophthalmos of the left eye disappeared in fourteen days, and vision was 0.4 in the right and 0.5 in the left eye; a month later vision was normal in the left eye. Slight exophthalmos and ptosis remained in the right eye. A corneal ulcer which developed in the right eye also disappeared during this period. Vasiutinsky states that this is the first instance of cure in a case of thrombosis of the cavernous sinus which he has observed among his 25 patients.

O. SITCHEVSKA.

PENICILLIN THERAPY IN OPHTHALMOLOGY. M. KRASNOV, Vestnik oftal. **25**: 9, 1946.

Krasnov analyzes the bacterial and chemical action of penicillin and the methods of its application. American penicillin was used with good results in an army hospital in cases of exudative uveitis, iridocyclitis and endogenous iritis. Intramuscular injections only were used daily, the total dose during a course of treatment ranging from 100,000 or 200,000 units to 2,000,000 units. A patient with syphilitic iritis responded well to treatment with 1,000,000 units of penicillin administered within ten days. Vision was improved from 0.02 to 1.0. Herpetic keratitis did not respond to treatment. A patient with severe intrabulbar neuritis of the optic nerve exhibited spectacular improvement in vision, from finger counting to 0.9, within a few days. Sulfonamide drugs used previously had had no effect. In a case of bilateral syphilitic neuritis penicillin therapy produced no improvement and the Wassermann reaction of the blood remained positive. Subconjunctival

injections of penicillin were accompanied with pain after the injections. Krasnov states that more clinical experience is necessary in order to evaluate the effect of penicillin therapy.

O. SITCHEVSKA.

INTRAOCULAR INJECTIONS OF PENICILLIN. J. A. VAN HEUVEN, Nederl. tijdschr. v. geneesk. **90:** 1006 (Aug. 17) 1946.

Van Heuven used 2 cc. of a solution of penicillin sodium (10,000 units of penicillin per cubic centimeter) for the irrigation of the anterior chamber of the eye. One of the patients, aged 74, had undergone an operation for cataract, which was followed by infection and panophthalmia. The second patient, aged 19, had been hit in the eye with a tennis ball. In the second patient an ulcer had developed at the margin of the cornea and sclera with hypopyon. In both patients irrigation with the solution of penicillin was followed by disappearance of the hypopyon. In the second patient the irrigation of the anterior chamber was repeated in five days. This procedure was followed by great improvement.

J. A. M. A. (W. ZENTMAYER).

Society Transactions

EDITED BY DR. W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Brittain F. Payne, M.D., *Chairman*

Milton L. Berliner, M.D., *Secretary*

Jan. 20, 1947

INSTRUCTION HOUR

Effects of the Atomic Bomb on the Human Eye. COLONEL J. E. ASH, Director, Army Institute of Pathology, Washington, D. C.

Information is to be cleared through the Surgeon General's Office at a later date.

REPORT OF CASES

Rheumatoid Involvement of the Extraocular Muscles. DR. J. B. LISMAN.

A case of recurrent diplopia is reported. The condition was observed in a white girl from the ages of 9 to 16 years.

The diplopia was due to paresis of individual extraocular muscles. Between attacks the diplopia was always overcome by fusion. A total of seven major episodes occurred between 1940 and 1947. During an acute attack, one could map out double vision due to paresis of an individual muscle, with diplopia increasing in the field of action of the muscle. There was pain on looking into the field of the paretic muscle, with tenderness on palpation over the muscle. There was repeated association of ocular involvement with migratory polyarthritis, erythema nodosum, fever, rapid sedimentation time and subcutaneous nodules. Biopsy of a specimen of an excised subcutaneous nodule from the scalp revealed nonspecific fibrosis. There were no cardiac abnormalities. It appears that the external ocular muscles were affected by a transitory inflammatory process, the cause of which was the same as that producing the generalized rheumatoid syndrome.

DISCUSSION

DR. HAROLD WHALEY BROWN: I saw this patient last July, just after the acute phase of her last attack of polyarthritis had subsided. Her only ocular abnormality at that time was vertical diplopia when looking to the left. The diagnosis of paresis of the right superior oblique muscle was made from the observation of right hypertropia with the greatest deviation (12 prism diopters) with the eyes down and to the left. This deviation was apparently not due to any pain or mechanical interference of the action of the right superior oblique, as there was no discomfort or demonstrable limitation of action of the muscle in extreme depression of the right eye in the nasal field.

Six months later (Jan. 3, 1947) measurement of the muscle balance with the screen cover test in the six cardinal fields of gaze

revealed a balance for near vision. There was no spontaneous diplopia in any field, either with or without the red glass.

In my experience paresis of an individual muscle is an uncommon finding in cases of rheumatic fever or chronic arthritis. In cases of episcleritis there is usually no muscular paresis unless Tenon's capsule is involved. (The edema and pain on movement of the globe are the evident cause of the underaction of the ocular muscles, and usually more than one muscle is involved.) It is difficult for me to account for the attacks of transient paresis of an individual muscle of a few weeks' duration on the basis of actual inflammation of the muscle, its sheath or its tendon. I think the transient paresis can be better explained on the basis of a toxic myositis, resulting from the general systemic condition of the patient.

I should like to ask Dr. Lisman whether repeated determinations of the sedimentation rate were made and, if so, what the findings were. Also, was there always an area of tenderness or swelling over the attachment of the muscle preceding each attack of paresis of an individual muscle?

Dr. J. B. LISMAN: The sedimentation rate was high during many episodes. As to palpation over the affected muscle, I did not try it in the earlier years, since the diagnosis was rather obscure. In the past few years pain has been a constant symptom. The pain is not diffuse but seems to be localized over the affected muscle.

Surgical Exposure of the Inferior Oblique Muscle. DR. WALTER F. JOHNSON.

I am suggesting a new and direct surgical approach to the insertion of the inferior oblique muscle, which eliminates the dangers of seeking the muscle by making blind sweeps with a muscle hook and permits operation at the insertion to be carried out more easily.

This paper will be published in full in a future issue of the ARCHIVES.

DISCUSSION

DR. RAYMOND EMORY MEEK: How and where to open the conjunctiva is important. On two occasions I have seen students casually pick up the conjunctiva to make the conjunctival incision in an operation on one of the rectus muscles, and then sweep a hook back and find there was no muscle there, simply because they had picked up the muscle with the conjunctiva and severed it from its insertion. After the incision is made in the conjunctiva, Tenon's capsule must be picked up and opened. Dr. Johnson uses a suture in the corneoscleral margin to pull the eye toward the nose and another suture looped around the inferior oblique to isolate the muscle. Greater adduction can usually be accomplished by using a squint hook under the external rectus muscle. Speaking of the conjunctiva reminds me of the time Dr. Wheeler came to the anatomy laboratory when I had a class at New York University. He showed us how to pick up the conjunctiva. He said the Lester forceps were to be used as a probe, passing one blade under the tissue and then pressing down the other blade; the tissue is then carefully picked up without getting too much conjunctiva.

Dr. Johnson's approach has been used on both the lateral rectus and the inferior oblique muscle. In making a recession of 3 to 4 mm., we make the conjunctival incision at the inferior border of the external rectus muscle, but in making a longer recession it is usually best to make the conjunctival incision 1 mm. below the lower border of the external rectus muscle. The insertion of the inferior oblique muscle is 1 to 3 mm. above the lower border of the external rectus muscle.

PAPER OF THE EVENING

Complications in Operations for Retinal Detachment. DR. J. A. VAN HEUVEN, Utrecht, Netherlands.

There is no reason to dwell on the operative treatment of detachment, as this subject has been extensively considered by Weve in "Modern Trends in Ophthalmology." At this time I wish merely to emphasize some of his statements. In the examination, my colleagues and I prefer to employ indirect ophthalmoscopy, with an extra strong light source. We always make a drawing of the whole fundus. The strong source of light is at the same time used for localization of the retinal tear on the outer wall of the eyeball. An assistant watches the spot where the light strikes the sclera. [Other methods of localization were discussed.] A drawing of the fundus not only assists in making sure that no part of the retina is overlooked, but makes it possible to recognize different types of detachment.

There is a possibility that accommodation plays a role in the origin of a detachment. There can be little doubt about a forward movement of both choroid and retina in accommodation, owing to the contraction of the radial fibers of the ciliary muscle. Clinical observations indicate that in special circumstances this movement may promote tearing of the retina. After operation one may see hemorrhages, both from retinal and from choroidal vessels, the prognosis of which is often not bad.

The picture of detachment of the choroid, with its sausage shape and chocolate hue, is not rare. Prognosis is good. Stellate figures and strands in the vitreous form a serious complication. These strands may appear in cases of detachment with a hole in the retina which has already been treated surgically, in cases before the operation, in cases without a hole and, last, in cases even without detachment.

The general condition of the patient seems to play a dominating role. [The various therapies were discussed from this point of view.]

Tumors of the choroid can make the diagnosis difficult if they are accompanied with a large detachment. [The differential diagnosis of cysts was discussed, and, finally, detachment following inflammation, especially that of a tuberculous nature, was considered.]

DISCUSSION

DR. ALGERNON B. REESE: I understood Dr. van Heuven to say that the operation is not over until the retina is back in place. The matter was then dropped, but I should like to have him carry the thought a little further, if he will. I should like to ask him how he takes care of detachments in aphakic eyes, in which it is difficult to see the periphery of the fundus. I am humbled by what Dr. van Heuven has said about his operative results because of my experience with

scleral resection. In 10 cases in which I did the operation I have not had a "cure." It depends, of course, on what one means by a "cure," and I feel that a cure is obtained only when the retina is back in place and vision is improved. This does not mean that the vision has to be normal. What do you consider a cure, Dr. van Heuven?

DR. FRANK A. VESEY: I should like to ask Dr. van Heuven whether he considers the reason for detachment of the retina purely mechanical and whether he thinks that as soon as holes develop anywhere in the retina detachment is due to ensue. If he holds the latter belief, how does he explain the fact that a traumatic hole of the retina in itself practically never causes detachment in an otherwise normal eye? How does he explain the failure of detachment of the retina to develop in senile eyes with large macular holes? Almost daily one sees a case of rupture of the retina without perforating wound of the sclera in which detachment does not occur. As an example of the effect of a perforating wound, I should like to cite a case I saw in the Army. During bayonet exercise a young soldier sustained a wound 3 mm. in length about 12 mm. from the limbus. The wound was properly closed, and he recovered without infection. Afterward, the scar in the retina was clearly visible, and detachment never occurred. I also shall take the liberty to ask Dr. van Heuven whether he studies the peripheral region of the fundus in senile eyes as carefully as he does in eyes with retinal detachment, and whether in such eyes he has ever seen severe cystic degeneration with multiple holes at the periphery without detachment.

DR. HENRY MINSKY: I was much impressed with the speaker's emphasis on the role of accommodation in the production of a hole in the retina! I believe he is on the right track, for in no other way can one explain the tear except through a pull by the ciliary muscle. During the last four or five years I have included, in addition to the regular diathermy, cyclodiathermy of that sector of the ciliary body in the region of the tear, and I believe the results have been a little better than before.

DR. J. A. VAN HEUVEN: In answer to Dr. Reese's question: I said that we do not consider the operation finished before the retina is back in place. That should be the aim—I do not say that we always accomplish this objective. There is always a certain number of cases in which we are doubtful about the result from the beginning. I think the prognosis is much better if the retina can actually be reattached at the moment of operation; we then feel more or less safe. However, even though we succeed in getting the retina attached, it will not necessarily remain in position permanently; but at least it has a better start.

The cases of aphakia still present a difficult problem, especially for the examination. We now and then examine the aphakic eye ophthalmoscopically with use of a contact glass, which makes it easier to see the periphery of the fundus.

It was also asked what I mean when I say a case is "healed." That is a good question, because there are statistics published in Europe which may be too optimistic. I do not mean that these statistics are unreliable; rather, I am saying that frequently an eye has been considered "healed" when the retina was reattached, without taking into

account the function. The patient, of course, is interested in what he sees; he does not mind much whether his retina is attached or not if he is blind. In these statistics there are a considerable number of eyes called "healed," meaning anatomically healed, but the patients are blind. This has given rise to international statistical confusion.

There are cases in which a hole in the retina is not followed by detachment, at least by full detachment, of the retina. I noted that foveal holes often give rise to a limited detachment. It is quite true that in elderly people one may find cysts, often taken for holes, or scars of local choroidoretinitis in the periphery which are not followed by full detachment. We have always thought this natural, because scar formation and healing have followed, similar to that which we try to achieve with diathermy or other therapeutic methods. One sees cases in which the new rim of the retina has been firmly attached to the wall of the eye. In other words, healing can occur spontaneously. After traumatic puncture of the eye one occasionally sees the same thing; but as a rule we find that after a traumatic lesion of the back of the eye a detachment occurs. When, for example, a piece of iron is extracted by means of a magnet, we have made it a rule to apply surface coagulation at the place of perforation and at the place where it has been extracted, if an opening in the sclera must be made.

I am much interested in the relation of accommodation and detachment of the retina. I omitted part of my paper on this aspect of the problem. I have seen cases of high grade myopia in which after strong use of the accommodation a tear developed in the retina. Our attention was drawn to this possibility because we observed it in 2 or 3 patients. However, it is still difficult to say whether the detachment occurred because of extraordinary use of their accommodation.

DR. ALGERNON B. REESE: If the retina is not back at the time the operation is completed, do you carry out further drainage of the subretinal fluid, or inject air or fluid into the vitreous cavity?

DR. J. A. VAN HEUVEN: I do not inject air or fluid, but try to suck out as much of the subretinal fluid as possible, and I place the patient in a position with the area operated on as low as possible.

Brittain F. Payne, M.D., *Chairman*

Milton L. Berliner, M.D., *Secretary*

Feb. 17, 1947

INSTRUCTION HOUR

Local Anesthesia in Ophthalmology. DR. WALTER S. ATKINSON, Watertown, N. Y.

The importance of producing complete anesthesia as painlessly as possible before beginning the operation was emphasized.

Pentobarbital or phenobarbital and acetylsalicylic acid are the sedatives preferred. Comparison of several local anesthetics for topical use showed tetracaine hydrochloride to be preferable. Procaine hydrochloride is still considered the safest and best for injection.

Of the various methods of producing temporary paralysis of the orbicularis muscle, O'Brien's is considered the most efficient. The

addition of 25 per cent alcohol for operations on the globe when there is a facial "tic" with spasmodic contractions of the orbicularis prolongs the paresis to from seven to ten days.

Retrobulbar injection of a solution of procaine hydrochloride within the muscle cone for operations on the globe, particularly cataract extractions, produces complete anesthesia of the globe, hypotony, a deeper anterior chamber and paresis of the rectus muscles. In cases of acute glaucoma the pain is relieved immediately, the intraocular pressure is lowered and the miotics are more effective.

For dacryocystorhinostomy, block of the infratrochlear and infraorbital nerves in the canals or in the sphenomaxillary fissure and a nasal pack produce good anesthesia.

With instillation anesthesia, infiltration or lid block, complete anesthesia of the lids may be obtained.

REPORT OF CASES

Cranial Epidermoid (Cholesteatoma) with Erosion of the Orbital Roof. DR. ISADORE GIVNER and DR. HENRY WIGDERSON (by invitation).

Primary cranial epidermoids originating in the diploe of the flat bones of the skull cause expansion of the overlying bone, with typical roentgenographic findings. Cushing observed that of 2,004 tumors of the brain only 0.06 per cent were epidermoids. Of 188 cranial epidermoids reported to date, 33 were of the diploic type, to which the one reported here belongs.

M. N., a man aged 84, first presented himself on Oct. 22, 1946, complaining of inability to open his left eye, due to pressure of a mass in the left frontal region, which, although present all his life, had grown considerably in the past three years. The mass was the size of a small apple, fluctuated and transmitted the intracranial pulsation. On deep palpation around the edge of the mass, the edge of the bone could be felt, suggesting absence of the frontal bone. On raising the left upper lid with the finger, one noted no elevating action of the eyeball. An immature cataract was present; the eye was otherwise normal.

On Dec. 4, 1946, with local anesthesia, the cyst was removed; a portion of the capsule adherent to the dura was left. The cyst contained 75 cc. of yellowish, oily, fluidlike content. The bony defect (7 cm. in diameter) included not only the frontal bone but the roof of the orbit as well.

When the patient looked up, the fascial roof of the orbit was retracted. When the patient looked down, the fascial roof moved upward into the cranial cavity.

The postoperative course was uneventful. Examination on February 4, 1947 showed equal palpebral fissures and return of function of the elevator muscles of the left eye.

DISCUSSION

DR. HENRY WIGDERSON: It is interesting to point out the typical roentgenologic appearance of the epidermoid. This is really pathognomonic, and when one has seen it there is never any doubt about the

diagnosis again. In most instances the epidermoid is benign. I have read of 1 case of epidermoid of about sixty years' duration in which the patient had been operated on a number of times during a period of thirty to forty years. Autopsy revealed carcinoma. The present case is the only one I know of in which the epidermoid appears to have undergone a malignant degeneration. In this case it would have been fairly easy to remove all the cyst wall, but, on account of the patient's age, I did not want to attempt to do this, and perhaps tear the dura. Often it is difficult to remove all the cyst wall because the tumor is of long standing and grows into the foramens and fissures of the skull. I had a case in which the tumor not only involved all the roof of the orbit but grew down into the middle fossa and involved the carotid artery. I did not try to touch the capsule at the base, and within a year and a half the patient had a recurrence of the tumor.

Dr. Alfred Kestenbaum: Dr. Givner and Dr. Wigderson reported an interesting phenomenon. In their case the orbital roof was replaced by a movable membrane. This membrane showed a definite upward movement when the eye looked downward and a downward movement when the eye looked upward. In order to understand this phenomenon, I studied the physiologic movements of the lids associated with the movements of the eyes. The position of the eyelid was observed by means of a pencil, its tip being placed on the lid. Normally when the eye looks upward, the lower lid bulges forward to a certain degree and the upper lid recedes; when the eye looks downward, the lower lid recedes and the upper lid bulges forward. Let us call the plane touching the upper and the lower lid at the middle of the lid margins the "tangential lid plane." This tangential lid plane is in a vertical position in gaze straight ahead; in looking upward the tangential plane is inclined backward; in gaze downward the tangential plane is inclined forward. Hence, the tangential lid plane consistently stands more or less parallel to the basis of the cornea. In the case of Givner and Wigderson these physiologic movements of the lids occurring with the ocular movements were observed in the normal eye, whereas they were definitely diminished on the side of operation.

The mechanism of the described comovements of the lids is apparently due to two factors: First, Tenon's capsule, which moves with the eyeball, is intimately connected with the neighboring orbital tissue and indirectly with the eyelid. In gaze upward, for example, retraction of the upper part of Tenon's capsule will result in retraction of the upper lid. The connection between eyeball and eyelid is still strengthened by so-called inhibitory ligaments and by smooth muscle fibers. Therefore each movement of the cornea upward or downward will be accompanied with retraction of the upper or the lower lid. Second, as long as the orbital wall is intact, retraction of the lower part of the orbital tissue in gaze downward will, owing to a kind of unchangeable hydrostatic pressure, cause a bulging forward of the upper part of the orbital tissue and hence of the upper lid; inversely, retraction of the upper part of the tissue in gaze upward will result in bulging forward of the lower lid. The second factor, however, will be effective only as long as the orbital wall is intact. If, as in the case

reported, a movable membrane is substituted for the orbital roof, this membrane will be able to yield instead of the upper lid. In gaze downward the membrane will therefore bulge upward; in gaze upward the membrane will be pulled down into the orbit. This explanation agrees with the mentioned fact that the comovements of the lid were normal on the unaffected side but were diminished on the diseased side.

Later, I observed another case in which a part of one orbital roof had been removed surgically. Again, examination revealed that the described physiologic accompanying movements of the lids were normal on the healthy side, whereas they were definitely diminished on the side of operation. Disturbance of the physiologic accompanying movements of the lids seems, therefore, to be of significance in cases of defect of the orbital wall. However, more cases will have to be studied before this sign can be considered infallible.

DR. JOSEPH IGERSHEIMER: As far as I know, only 1 case of epidermoid of the skull involving the eye has been reported in the ophthalmologic literature. As such cases are so rare, I wish to mention a case which I have been watching for several years. A colleague, aged 47, came to my office in February 1942, with the complaint that his right eye had been protruding for several days and that he could not see as well with this eye as with the other one. He said that for thirty years he had felt that something was wrong in the right frontal bone temporally and above the orbit. Within the last few weeks he had noticed growth and progressive protrusion of what he had always considered an exostosis in this region. In addition to exophthalmos and a certain diminution of vision, he noted edema of the right upper lid. This edema disappeared in a few days. There was a slight papilledema. The roentgenogram showed the characteristic Cushing sign (decalcification in the temporal part of the frontal bone). The diagnosis of cholesteatoma (epidermoid) originating in the diploe was made. He was operated on a month later, and the diagnosis was confirmed. The tumor was restricted to the anterior fossa. The bone of the orbital roof was extensively eroded. He recovered fully after the operation, except that the exophthalmos did not entirely disappear. There was recurrence a year later, demonstrated by an increase in exophthalmos, headaches and edema of the lid. The surgeon (Dr. Gilbert Horrax) decided to wait and see whether the symptoms disappeared, and they did. Several years later the patient again had sensitivity in the region of the tumor and the feeling of increasing exophthalmos, but these symptoms also diminished shortly. The fluctuating character of the symptoms in this case is of interest; in this connection, I should like to mention that even before operation he showed a relative bitemporal hemianopsia, which disappeared before the operation was performed.

Unilateral Exophthalmos Due to Meningioma of the Middle Fossa.

DR. HAROLD KIRSHNER (by invitation).

The causes of unilateral exophthalmos may be divided into five groups: vascular, inflammatory, tumor, trauma and general disease. These include a diversity of pathologic conditions, among which arteriovenous aneurysm, varicosities of the orbital veins, cavernous angioma,

periostitis of the orbit, thrombosis of the cavernous sinus, trauma, xanthomatosis, purpura, hemophilia and orbital tumor are most frequently encountered.

A case of unilateral exophthalmos of the right eye due to pterional meningioma *en plaque* was presented. The space-occupying character of the tumor and the resulting hyperostosis of the sphenoidal ridge, the greater and lesser wings of the sphenoid, the roof of the orbit and the pterional region explain the mechanism of the exophthalmos, which measured 3 mm. The exophthalmos was not reducible. Except for diminished corneal sensitivity, no other involvement of the third, fourth, fifth or sixth cranial nerve was noted. In view of the patient's age (53) and vascular status, neurosurgical operation was deferred.

DISCUSSION

DR. JAMES W. SMITH: I should like to review at this time a case similar to Dr. Kirshner's which I presented before this Section eight years ago. Dr. Kirshner has already referred to the classic features of the syndrome enumerated in my paper. My patient, a woman aged 53 with more extensive involvement than in Dr. Kirshner's case, was operated on by Dr. Leo M. Davidoff on Feb. 6, 1939. The slow growth of this type of meningioma was demonstrated in my case. I have had an opportunity to examine the patient repeatedly since Dr. Davidoff's operation. Since one eighth of the bony hyperostosis could not be removed with safety, a recurrence was expected. For three and a half years ophthalmic examinations revealed nothing significant. The exophthalmos has progressed about 1 mm. a year in the past four years. Vision is now reduced from 20/20 to 20/30+. The field of vision and the fundus are normal. The patient's only complaint is epiphora, when outdoors, which is controlled by a moist chamber spectacle. I suggested a plastic device for the lid to narrow the palpebral fissure, but the patient prefers to wait until another neurosurgical procedure is indicated, which she feels will again reduce the exophthalmos. Dr. Davidoff, however, feels that, in view of the extremely slow advance of the recurrence, the patient, who is 62, should not be subjected to another formidable operation until definite pressure on the optic nerve is evident.

DR. TOMAS R. YANES, Habana, Cuba: It may be interesting to cite a case my colleagues and I had in Cuba about six months ago, that of a woman aged 40 who had exophthalmos due to a meningioma of the middle fossa. She decided to come to the States and to be operated on in Dandy's clinic. Dr. Dandy was very ill and died two or three days after her arrival; so the operation was done by an assistant, whose name I do not know. The surgeon told the family that the chances of death at operation would be about 8 per cent and that if she were not operated on she might live five, six or eight years longer. Considering her age, she decided to be operated on, and she is now perfectly well.

DR. HAROLD KIRSHNER: The only unusual feature of this case is the fact that the changes in the optic nerve appeared late, i. e., in the fifth or sixth year. In the classic syndrome of meningioma *en plaque* there are no changes in the optic nerve, at least not in the early phases of the disease.

PAPER OF THE EVENING

Beta Radium in Treatment of Corneal Lesions. DR. A. D. RUEDEMANN, Detroit.

Growths on the Lid.—Large, infiltrating growths suggestive of carcinoma should be excised and treated with gamma rays of radium or with roentgen rays. When the growth is small, shallow or difficult to excise, beta rays of radium are the therapeutic choice. Although beta rays are soft, only one-half a given surface dose is absorbed in 2 mm. of tissue, and all minor lesions are easily and successfully treated. Small marginal growths, to which it is difficult to apply gamma or roentgen rays, can be given one or two applications, and they will be destroyed with little, if any, loss of lashes. Beta radiation can be used as well for small angiomas in babies. The time of application is short, and, with a simple holding technic, no anesthetic is required. In treatment of other cutaneous lesions around the eye, use of beta radiation is suggested, as it leaves no discoloration of the skin or depigmentation and there is no tendency to a deep bronzing effect.

Vernal Catarrh.—If the polyps are large and long standing and fibrotic, excision is indicated. Gamma radiation is then used. The eye must be carefully screened with lead plates, the lid everted and the applicator directly applied. This therapy gives prompt relief but may need to be repeated. Cold compresses and irrigation minimize edema and reaction. Large polyps having been excised or reduced in size, beta radiation can be directly applied to the individual polyps, especially those along the superior aspect of the tarsus. Mild catarrhal lesions may be treated with spray radiation. The effect is prompt and lasting. Individual polyps and small areas are easily reached. Except in urgent cases, in which use of gamma radiation is advisable, beta radiation is preferable for children, as it can be more easily and more quickly applied. The end results are similar. A long applicator to evert the lower lid and a double length tongue blade are safety aids in therapy.

Conjunctival Lesions.—Small lesions on the bulbar conjunctiva can be treated directly with beta radiation. Small, superficial growths require but one application, rarely two, with direct contact. Vascular growths are more resistant but can be treated with direct contact. Spray radiation is of value. Enlargement of the caruncle, enlarged pingueculae and beginning pterygium are well treated by direct application. Small pseudopterygiums are treated by direct application. Large, true pterygiums and pseudopterygiums are treated by a combination of spray radiation over the corneal portion and direct application along the limbal portion. Recurring pterygiums having interstitial vascular trunks require contact radiation over most of the extent to eliminate deep vascularization. Marginal carcinoma is satisfactorily treated with direct radiation contact.

Cornea.—Deep and superficial keratitis, due to tuberculosis, syphilis or undulant fever, are known to be successfully treated with radiation. Other treatment should be directed at the etiologic factor. Corneal nebulae following ulcer, trauma or allergy are usually cleared, with little residual scarring. Maculas of the cornea can be substantially reduced in area and may be sufficiently cleared to enable the patient to carry on without corneal transplantation. Vascularized leukoma can be treated

so that hazard of vascularization to a corneal transplant is lessened. Corneal transplants with a tendency to vascularization can be treated to prevent complete vascularization. Diminution in the area of corneal scarring and reduction in density are major factors in beta radiation therapy.

Treatment with Beta Radiation.—1. Treatment is either with direct contact or with spray radiation. 2. The time of treatment varies according to the type of lesion, strength of tube and reaction to treatment. 3. The time factor is important in irradiation of corneal lesions—it is difficult for children and some adults to hold still longer than twenty-five seconds. Use of a stronger tube with a short time factor is advisable. 4. Age is not a factor; beta radiation treatment has been given patients 6 months of age. 5. The only discomfort to the patient is his own fear. 6. As a measure of economy a number of patients should be arranged for treatment at one time. This cuts the cost per patient and saves time. 7. Separate record cards are kept for instant reference.

Summary.—There is a twofold hazard in the use of radiation therapy: the danger to the patient—which lies in an overdose, in faulty application and in a severe reaction, and that to the ophthalmologist. The value of radiation therapy should be stressed because of its usefulness in so many conditions previously supposed to be incurable. The use of roentgen ray or gamma or beta radiation offers a valuable adjunct to therapeutics in ophthalmology.

To date, no definite evidence of injury to the eyes from beta radiation has been found. In several cases of severe corneal leukoma excessive treatment with beta radiation produced small perforating ulcers and enucleation was necessary. Roentgen and gamma radiation therapy may produce cataract and injure the retina permanently. It is possible to use the various forms of radiation in a single case. Care must be taken to allow for cessation of reaction if such a procedure is indicated. Photographs and drawings are valuable aids as progress notes.

DISCUSSION

DR. HAYES MARTIN: Dr. Ruedemann's presentation has been interesting to me, because it is my belief that if radiation therapy is to be used for any given disease or in any given part of the body it should be applied by specialists in that disease or in that part of the body rather than by therapeutic radiologists, who purport to treat all diseases anywhere about the body with radiation. It is encouraging that ophthalmologists themselves, such as Dr. Ruedemann, are beginning to apply radiation therapy for diseases of the eye, because out of their experiences, successes, failures and disappointments (for I am sure they are going to have many disappointments and complications which they do not as yet expect) will come some real knowledge of the possibilities of radiation therapy in these diseases.

Not being an ophthalmologist, I do not know enough about the disease conditions which Dr. Ruedemann demonstrated in slides to offer any worth while comment. I do not know whether these diseases could possibly have been better treated by methods other than irradiation, but I think that a final decision as to the indication for treatment of these diseases by irradiation rather than by other methods must depend on the natural course of the disease. That is to say, can Dr. Ruedemann

be certain that his short term results justify the hazard of the late sequelae of irradiation?

In this connection, I think it well to keep in mind that effects of radiation are always destructive. Radiation therapy is not a tonic, nor does it have wholesome effects anywhere about the body. It is always destructive in its action on normal tissues. Even though the effects of radiation are undesirable, they are in many cases preferable to the disease for which the radiation is given. The clearest example of this is cancer, the significance of which is almost always more serious than the complications of radiation therapy.

It is well to keep in mind that the late effects of radiation never disappear; they always become worse as time goes on. The untoward effects of radiation are more serious five years after application than after one year; they are worse after fifteen years than they are after ten years, and sometimes the most serious sequelae of therapeutic radiation do not become evident for thirty or forty years—in most cases long after the radiation therapeutist is dead and gone.

When one goes back twenty-five or thirty years in the history of radiation therapy, especially back to the early twenties, one will find that almost every disease afflicting human beings has at one time or another been treated with radiation and glowing results have been reported. In general, few such ideas persist today. With many diseases in which there may have been a favorable response to radiation at first, it has been found on mature consideration that older, simpler methods, when revived, were superior. In other, self-limited, diseases, it was found that the clinical course was not actually influenced at all by radiation and that remission or recovery took place just as quickly without treatment. In still other diseases for which radiation has been enthusiastically advised, the sequelae have been disastrous, and sometimes fatal.

From Dr. Ruedemann's presentation, I get the impression that he believes the response to radiation is practically always favorable and that he has had few, if any, failures and no untoward sequelae. I cannot understand how there could be no complications or sequelae.

For lack of time, I cannot comment on many of the complications following radiation therapy—take as an example one of the most common ophthalmic sequelae, radiation cataract. I gathered from Dr. Ruedemann's paper that no radiation cataract had developed in his cases. Over the past twenty-five years at Memorial Hospital my colleagues and I have seen a large number of radiation cataracts, and they seem to follow small, as well as large, doses. We know of no way to prevent them except to let no radiation whatever hit the lens; in this connection it seems strange that Dr. Ruedemann made no mention of protective shielding. But, after all, so far as radiation cataract is concerned (and other complications of irradiation), the risk is justifiable provided that the disease for which the radiation is given is in itself more serious than radiation cataract (or other sequelae). In my opinion, radiation is not otherwise justified.

I listened carefully during Dr. Ruedemann's presentation for some statement regarding dosage, and I was disappointed that he made no mention of it. To say that the patient had so many applications of "beta radiation" is not enough; I should like to ask Dr. Ruedemann,

in closing, to tell us something of the dose used. The radon applicator which he mentions was devised about thirty years ago. I well remember its use at Memorial Hospital over twenty-five years ago; so there is nothing new about this phase of the subject. The dose of such a radon applicator is usually stated in millicurie minutes; I should like to know Dr. Ruedemann's dose in millicurie minutes for the various lesions he describes.

I trust that you will not consider my discussion too critical and destructive, for I do not mean it in that way; but I think it is well to utter a word of caution, since it seems to me that Dr. Ruedemann's attitude toward the problem is a little too optimistic. Radiation is not like penicillin, with which there is no after-effect whatever. It is not even like many toxic drugs of which the untoward after-effects are short lived and from which recovery can be complete. The untoward after-effects of radiation are permanent; they always persist and continue to grow worse.

In closing, I wish to repeat what I said in the beginning, that ophthalmologists themselves should continue to administer radiation wherever indicated for lesions about the eye. The ophthalmologist not only should apply the radiation himself, but should also decide on the dose. He should take on himself complete responsibility for the results, reporting untoward sequelae, and failures as well as successes. He cannot ethically transfer to a physicist or a radiologist all the technical phases of the treatment. Only by the application of such principles will progress be made in radiation therapy.

DR. RAMÓN CASTROVIEJO: We are grateful to Dr. Ruedemann for his presentation on the treatment of corneal lesions with beta radiation. He seems to be somewhat discouraged with the results he has been able to obtain with keratoplasty and points out that surgeons who carry out corneal transplantation will have many disappointments. He is right in this statement. The man who undertakes to perform corneal transplantation sooner or later finds that there is no other surgical procedure in ophthalmology which gives so many complications. These complications often depend not on the ability of the surgeon to perform the operation but, rather, on the reaction of the patient's eye to the surgical trauma. There are transplants which, after a few weeks of transparency, become cloudy on account of such complications as uveitis or vascularization of the graft.

Keratoplasty is not indicated in all cases of corneal opacities and is contraindicated in many cases of corneal deformities, for there are eyes which do not lend themselves well to this type of operation. This is the negative aspect of keratoplasty. On the other hand, if the conditions have been carefully selected, such as central leukoma, interstitial keratitis and keratoconus, the well trained surgeon may expect a transparent graft in as high as 80 per cent of cases and great improvement in vision.

In Dr. Ruedemann's treatment of corneal lesions with beta radiation, he seems to have obtained the best results in cases in which infiltration rather than fibrosis was present in the cornea. I am afraid that where there is fibrosis beta radiation therapy will not be satisfactory. In cases of densely vascularized leukoma, use of beta or roentgen radiation properly administered will be a good method of

treatment after operation, provided the irradiation is done shortly after the surgical procedure has been carried out. If the irradiation is instituted immediately after surgical treatment, the capillaries, which are responsible for recurrence of corneal vascularization, can be easily obliterated, while they are extremely radiosensitive, with a minimum dose of beta or roentgen radiation. On the other hand, if too long a time is permitted to elapse after operation before the application of radiation, higher doses of beta or roentgen radiation will be necessary to obliterate the vascularization and diminish the fibrosis, with possible subsequent damage to the eye. Dr. Martin has already pointed out not only the immediate but also the late complications in tissues which have been exposed to high doses of radium or roentgen rays.

I have not had any experience with beta radiation but have treated a substantial number of cases with roentgen rays. In the future I intend to use both beta and roentgen radiation in eyes with similar lesions in order to evaluate their respective efficacy. At present my colleagues and I do not regard as adequate the dose of roentgen radiation which we have been using after surgical measures on the cornea. We are still comparing results of treatment with different doses in order to arrive at the minimal optimum dose. The 600 r which was the standard dose used in many cases after keratectomy and keratoplasty with a tendency to vascularization was found to be insufficient. In some cases we are now giving as much as 1,200 and 1,500 r. I should like to single out for discussion some of the cases presented by Dr. Ruedemann, but limitation of time will not permit me to do so. Dr. Ruedemann has been on the pessimistic side in appraising the results of corneal transplantation in treatment of corneal lesions; on the other hand, I feel that he has been somewhat too optimistic in evaluating the results of beta radiation in treatment of these lesions. Further study of both keratoplasty and irradiation will determine more accurately the indications for each method in the treatment of corneal lesions.

DR. VITO LA ROCCA: I should like to ask whether Dr. Ruedemann has used beta radiations with lesions of vernal catarrh and whether there has been any recurrence of vegetations through the scar.

DR. A. TORRES-ESTRADA, México, D. F., Mexico (translated literally by Dr. Ramón Castroviejo): In Mexico, my colleagues and I have been using gamma radiation for some time in the treatment of various corneal lesions. It is most often used after dacryocystorhinostomy, one of the most frequent causes of failure of which is the formation of keloids. We can prevent the recurrence of epiphora by the application of beta radiation seven days after the operation, particularly if lacrimation begins to appear. We also use roentgen radiation in the treatment of some cutaneous growths, particularly on the borders of the eyelids. Follicular conjunctivitis is best treated by two or three applications of beta radiation, which cicatrize without leaving any sequelae. In Mexico we have a great deal of vernal conjunctivitis, which is well treated with beta radiation.

Pterygium is treated in Mexico by special surgical procedures, the corneal apex of the pterygium being dissected from the cornea and Bowman's membrane preserved. The fibrous tissue of the body of the pterygium is dissected between the sclera and the epithelial layer. This fibrous tissue is excised, and the exposed area of the sclera is covered

with a conjunctival flap. The area of operation is treated with gamma or beta radiation. With this treatment we have few recurrences of pterygium; when they do recur, they are caused by faulty technic, faulty section of the pterygium or too extensive resection of corneal tissue.

Radiation is also employed in cases of keratitis and of keratoplasty when the transplant becomes nebulous; with keratitis particularly, the application of beta radiation seems to give good results.

Finally, of late we have been treating patients with advanced glaucoma with radiation, particularly secondary glaucoma due to intraocular hemorrhage. Eyes which should have been enucleated have been treated with radiation, with relief of the painful symptoms; and some eyes have thus been saved from enucleation. Because of this good result in treatment of glaucoma, we have used radiation in cases of hemorrhagic retinitis, with some resulting improvement. I cannot give much information on the technic or dosage of roentgen irradiation. Although the treatment has been given under my supervision, the technical details have been left entirely to an expert.

DR. A. D. RUEDEMANN, Detroit: I wish to thank Dr. Martin for his courteous discussion. I am a rank amateur in the treatment of ocular lesions with radiation. I can assure him I do not move without a roentgenologist, a technical man, beside me, for where the roentgenologists fear to treat should not be a fool's paradise for the rest of us. I can assure you my colleagues and I have had more bad results than successes. A number of corneas have perforated, as I have said before; some of the scarring has been made definitely worse, and I can give a longer list of conditions of the eye which one should not treat with radiation than of those one should so treat.

In regard to the dose, Dr. Otto Glasser, of the department of biophysics, estimates that for us; it is the amount of radon substance in the glass vial which gives the number of treatment seconds to make 250 to 300 millicurie minutes, and the treatment usually runs twenty seconds. In the case of a very thin scar of the cornea, it is possible to give it one-half, rather than a full, twenty second treatment; otherwise, the reaction is too severe. One can produce such an intense reaction that the scarring from therapy is as great as would have occurred originally. Therefore, we give the patient three ten second doses and wait three months. When the scar is very dense, we give the full treatment. In several instances we have tried twice the amount and have run into trouble; it was in these cases that we had the corneal perforations.

We have had recurrences of some of the vegetations in cases of vernal conjunctivitis, but they have not involved the entire lid, and we have been able to pick them up as individual plaques and treat them with the applicator. The amount of scarring of the lid is minimal rather than maximal, and the eye is fairly comfortable after one or two applications. In the case of large polypoid masses it is best to screen off the eye with lead, and we have always referred such patients to Dr. U. V. Portmann, of the department of therapy; he treats them and sends them back to us for the small marginal polyps and the individual polyps on the surface of the lid.

I am perhaps a little pessimistic about corneal transplantation, for I probably do not do it as well as Dr. Castroviejo—I am sure I do not—

perhaps I should remain pessimistic, so that those of us who are not so expert do not become too optimistic about the method.

The ill results of radiation therapy are often designated by the patient himself. If the patient is satisfied and can see well enough to do what he wants to do and carry on a job, he does not want operation, and I do not urge him into it.

I thank Dr. Castroviejo for the discussion of the radiation therapy being used in Mexico. Apparently ophthalmologists there are farther along than we are. There are certain things with which I do not agree. I do not believe that gamma irradiation should be used for recurrent pterygium. Again, it is advisable at the start of treatment to make good notes on the lens, for in most cases changes in the lens occur. In our early cases we made good notes on the lens, for we were interested in finding out whether this type of radiation would produce changes in the lens; to date we have not found any. That does not mean that we shall not have any, for I agree with Dr. Martin that the late sequelae might discourage one from using this method at all. Most patients with corneal lesions have a certain amount of change in the lens; they, therefore, have a double defect: the corneal lesion and the changes in the lens. We used to spray radiation over the capillaries, contact radiation, when we were treating a fairly dense scar or a good-sized vessel; I agree with Dr. Castroviejo that the treatment should be instituted within seventy-two hours after the operation on the cornea has been completed. In closing, I might say it is advisable for the ophthalmologist to have some one beside him who knows radium and roentgen ray therapy. Most ophthalmologists are not aware of the hazards connected with radiation treatment. There is a hazard if too much treatment is used; there is a hazard to the patient who is not treated properly, and there is a hazard to the user of the radium; radium is not something one can use without due regard to what is being used, for radium is a powerful substance.

Brittain F. Payne, M.D., *Chairman*

Milton L. Berliner, M.D., *Secretary*

Centennial Meeting, March 17, 1947

PAPERS OF THE EVENING

Past and Future Progress in American Ophthalmology. DR. DERRICK VAIL, Chicago.

The past one hundred years has seen the origin, development and full flowering of American medicine and, with it, of ophthalmology. Arising as an offshoot of European science and transplanted to the new and free environment of the United States, American ophthalmology has flourished like the "green bay tree." It will continue to do so unless stifled or smothered by political, bureaucratic manure.

At first a country cousin, a sort of ridiculed yokel, American ophthalmology has at last reached maturity and has become a distinguished and respected contributor to world ophthalmology. The period of rapid, self-sufficient and manly growth began after World War I and accelerated shortly before and during World War II. The

reasons for this are many. First, there is the heritage handed down by the early great ophthalmologists, most of whom were trained during the golden age of European medicine, which is perhaps forever gone; second, the growth and influence of our dynamic ophthalmic societies; third, the impact of the American Board of Ophthalmology on all teaching of ophthalmology in this country; fourth, the increasing interest of many foundations in ophthalmic research; fifth, the growing opportunities for postgraduate training in this country; sixth, the impressive increase and significance of native ophthalmic literary and scientific contributions; seventh, the development and maturity of American ophthalmic journals, and, finally, the vigorous quality of the leadership of teachers in the medical schools, hospitals and medical centers of the country.

Seed planted in such fertile and rich soil is certain to sprout and be fruitful if it is properly tended, pruned, cultivated and enriched. Our future progress in ophthalmology in America must be dependent on the continuance of our program of teaching, training and instruction, on the fostering of every plan for the dissemination of knowledge through scientific meetings, free discussions, books, journals, instruction courses and, particularly and above all, the example, precept and integrity of our leaders. Our progress can only be wilted, sterilized and vitiated if, by our folly, we succumb to political machinations, to harmful, misleading and self-aggrandizing publicity, and to propaganda which is alien to the free development of our science.

History of Ophthalmology in Canada. Dr. John A. MacMillan, Montreal, Canada.

A short introduction concerned the early history of medicine in Canada under the French and British régimes. This was followed by short biographies of the pioneers in the field of ophthalmology, including Henry Howard, Abner M. Rosebrugh, Richard Andrew Reeve, Frank Buller and Louis Edward Desjardins. Finally, the present endeavor to meet the demand for training in ophthalmology was discussed.

History of the American Board of Ophthalmology. Dr. S. Judd Beach, Portland, Maine.

The American Board for Ophthalmic Examinations was organized in 1915. This is now the American Board of Ophthalmology. Ten years later the next examining Board, that for the ear, nose and throat, was launched. The idea of examining ophthalmologists and of issuing a certificate to be required for admission to the special societies originated with Edward Jackson.

Before the organization of the Board for Ophthalmic Examinations, no standards for graduate training were in effect. Residencies were without systematic teaching. Graduate courses covered no specified subjects. No attempt was made to examine students to find out what they had actually learned.

Requirements, established by the Board, have compelled the standardization of such casual graduate teaching. The Board has also furnished the public with a list from which safe ophthalmologists may

be selected, for this has always been the objective of the examination. From this pioneer project has developed the entire system of examinations in medical specialties, now numbering fifteen boards, coordinated by the Advisory Board of Medical Specialties.

New York as an Ophthalmological Center. DR. BERNARD SAMUELS, New York.

The first ophthalmic institution in New York was the New York Eye Infirmary, now the New York Eye and Ear Infirmary, founded in 1820. For thirty-two years it was the only institution of its kind in New York. In 1852 the New York Ophthalmic Hospital was founded, and since then seven others have come into existence. Each of these hospitals was named and briefly described in chronologic order.

Ophthalmology in New York was discussed under the headings of surgery, teaching, research and literature.

Reference was made to the benefits ophthalmology has derived from the Academy.

The conclusion was reached that New York is the foremost ophthalmologic center of the world.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Burton Chance, M.D., *Chairman*

George F. J. Kelly, M.D., *Clerk*

Feb. 20, 1947

Syndrome of Paroxysmal Lacrimation When Eating Associated with Paralysis of the Lateral Rectus Muscle (Gusto-Lacrimal Reflex of "Crocodile Tears"). CAPTAIN FRANK C. LUTMAN, Medical Corps, Army of the United States (by invitation).

Paroxysmal lacrimation when eating, which is occasionally seen as a sequel to peripheral paralysis of the facial nerve on the same side, is an abnormal form of reflex tearing accompanying salivation. The explanation for the abnormal facial movements following peripheral motor paralysis of the facial nerve and other pathologic associated reflexes is also applicable here. From all cases now available, the evidence suggests that this reflex tearing is established either by some of the regenerated axons to the salivary glands becoming misdirected to the lacrimal gland or by single axons at the point of the lesion branching and innervating both the lacrimal and the salivary glands. However, the center for this reflex, if peripheral, remains obscure.

Two cases were described. In the first congenital unilateral paroxysmal lacrimation when eating was associated with homolateral paralysis of the lateral rectus muscle. In the second there were bilateral paralysis of the lateral rectus muscle and bilateral reflex tearing when eating. A review of the literature on lacrimation when eating revealed a third case of the syndrome, with similar bilateral involvement. In all 3 cases the condition was first noticed in infancy.

It was suggested that the pathologic syndrome in these 3 instances was produced by a pontile lesion, involving the nucleus of the abducent

nerve and the superior salivary nucleus of the facial nerve, or by a lesion of the nucleus of the abducens nerve and the genu internum of the facial nerve as it passes around the nucleus of the sixth nerve.

These 3 cases of paroxysmal lacrimation when eating associated with paralysis of the lateral rectus were presented as evidence of a new congenital neuro-ophthalmologic syndrome.

As treatment, if the reflex tearing can be interrupted by cocainization of the sphenopalatine ganglion, injection of alcohol into the ganglion offers a more permanent means of control. When indicated, the paralysis of the lateral rectus should be treated with tendon transplants from the superior and the inferior rectus muscles.

DISCUSSION

DR. ROBERT A. GROFF: Captain Lutman has presented an interesting and, so far as I am aware, a new syndrome when he adds to the familiar paroxysmal lacrimation during chewing movements a homolateral paralysis of the rectus muscle.

I became interested in the syndrome without the associated palsy of the lateral rectus muscle six or seven years ago, when I saw a patient who teared whenever he ate an apple. This patient gave a history of a facial palsy, from which he had completely recovered eight or ten months before. As Captain Lutman suggested in discussing the treatment of this condition, I, at that time, injected procaine into the sphenopalatine ganglion and produced temporary cessation of the phenomenon during the time the drug was acting. Since this procedure was successful, the second injection was made, using 0.5 to 1 cc. of absolute alcohol, and for about three months the patient was free from symptoms. After that he disappeared from the clinic, indicating perhaps that the difficulty did not return.

As to the association of the syndrome with palsy of the abducens nerve rather than of the facial nerve, I have had no personal experience. This syndrome is new, although I say it with hesitancy, not having reviewed the literature. I do not agree with Captain Lutman that the syndrome is caused by a central lesion. The tearing associated with lesions of the facial nerve has been rather definitely proved to be a peripheral reflex phenomenon. The possible pathways are the chorda tympani, the sphenopalatine ganglion, then through the zygomatic nerves to the lacrimal gland. It may be that in some way the fifth cranial nerve is involved, in view of the studies Dr. Lewey and I carried out in connection with the Marcus Gunn phenomenon in which we demonstrated autonomic fibers in the branches of the trigeminal nerve which, when stimulated, caused movements of the eyelid after the third cranial nerve had been sectioned several weeks before. For this reason, and because a central lesion would necessarily have to be minute to produce this syndrome without giving other neurologic signs, the entire phenomenon could better be explained on the basis of a peripheral perversion reflex. Furthermore, the fact that relief was obtained by injection into the sphenopalatine ganglion tends to support this view.

I wish to thank Captain Lutman for the privilege of discussing this interesting presentation.

DR. WALTER I. LILLIE: I assure Dr. Lutman he has been fortunate in seeing so many cases during his short career in medicine, for they are rare indeed. I was much interested in his paper, as I have only seen 1 case with paralysis of the lateral rectus muscle and lacrimation. I feel as Dr. Groff does, that it is a peripheral lesion. Personally, I do not see how one could conclude that there is a pontile lesion in the region of the nucleus of the abducent nerve where the knee of the seventh nerve passes around it. The pons is a small portion of the central nervous system. A lesion large enough to include both the genu of the seventh nerve as it curves around the nucleus of the sixth nerve and the salivary center would produce many contralateral motor and sensory disturbances, and that, to my knowledge, has not occurred.

CAPTAIN FRANK C. LUTMAN, Medical Corps, Army of the United States: With Dr. Lillie and Dr. Groff, I also believe that this is a peripheral reflex. A lesion of the pontile facial nucleus would produce peripheral facial paralysis, which, as I brought out in the paper, is a requisite for development of this type of paroxysmal lacrimation. As I understand Dr. Groff's contention, it would require two lesions to explain a combined paralysis of the abducent and the facial nerve. To my mind, it would be simpler to attribute this to a single lesion, and the point where the facial and the abducent nerve are closest together is in the pons.

However, as has been mentioned, this tearing is rather complicated, and I hope that in future cases a careful neurologic examination will be made.

Syndrome of Meningeal Fibroblastoma Arising from the Lesser Wing of the Sphenoid Bone: Analysis from an Ophthalmologic Standpoint. DR. ROBERT H. TRUEMAN.

The historical aspects of suprasellar and parasellar meningiomas were considered, with particular emphasis on the early observations of those arising from the sphenoidal ridge.

The terms suprasellar and parasellar were defined, with a description of the sphenoidal ridge. The anatomy of the region of the lesser wing of the sphenoid bone and the sella turcica was reviewed, and the pathologic picture of meningioma was discussed.

The syndrome of early meningioma of the lesser wing of the sphenoid bone was described as atrophy of the optic nerve, changes in the visual fields, exophthalmos, involvement of ocular motility, sensory disturbances in the distribution of the first and second divisions of the trigeminal nerve and involvement of taste and smell, all on the side of the tumor. Further changes in these symptoms and signs with growth of the tumor were mentioned, as well as the appearance of symptoms and signs of involvement of the pituitary gland and the frontal lobe and sensory and motor alterations.

The symptoms in 56 cases were analyzed to show that in 82 per cent of them the first symptoms to appear were ocular. The various forms were discussed.

The objective findings were then analyzed to show that in 91 per cent of cases there were changes in the fundus, in 86 per cent changes in the visual fields, in 43 per cent exophthalmos and in 38 per cent involvement of ocular motility. The findings other than the ocular signs were then discussed, including trigeminal paresthesias, signs of pituitary disturbance, involvement of taste and smell, signs referable to the frontal lobe and sensory and motor changes.

The roentgenographic picture was then discussed, with particular reference to the generalized and the localized changes, which include the erosions and the local and general hyperostoses.

The differential diagnosis of intraorbital tumors, intrasellar tumors, aneurysms, optochiasmic arachnoiditis, gliomas and craniopharyngioma was discussed.

In conclusion, the syndrome was redefined, with the early and late clinical findings.

DISCUSSION

DR. ROBERT A. GROFF: Dr. Trueman is to be congratulated on his excellent presentation. Certainly, he has clarified the syndrome since Dr. B. J. Alpers and I first called attention to it, several years ago.

The successful surgical removal of these tumors depends on early diagnosis, for the technical problem is then simplified greatly. The lesion is small, and, as a result, the opportunity of complete removal is much more favorable.

The picture presented here includes that in cases of late diagnosis. In fact, in some of the cases the diagnosis was made at a time when the tumor was inoperable. Let me state the early signs, which are sufficient to make the diagnosis. Early primary optic nerve atrophy or papilledema, a cut in the visual field on the temporal side of the affected eye and a sella turcica at the upper limit of normal size are all that is necessary to justify one in demanding encephalographic studies to verify the diagnosis.

When unilateral exophthalmos is developing in a patient, the possibility of meningioma of the lesser wing of the sphenoid bone should be excluded first. Roentgenograms of the skull will aid in the diagnosis, for, in my experience, the sphenoidal ridge shows definite thickening incident to tumor infiltration.

At operation these tumors present an interesting picture. The meningioma in this location is one of two types: a bulbous growth or an en plaque type, in which the tumor is flat and spreads over the surface of the dura. The latter type is usually associated with underlying infiltration of bone, which produces exophthalmos.

In order to cure the patient, it is necessary to remove the dura to which the tumor is attached. The bulbous variety offers a much less technical problem than the tumor which grows by spreading over the surface of the dura. The former, therefore, if not large, can usually be removed completely. The latter is almost never removed completely and therefore recurs. Since this type of tumor is of rather slow growth, it is usually several years before a second operation is necessary.

When the bone has been invaded by the tumor, it is practically impossible to remove all the involved bone because of the adjacent

structures—the internal carotid artery, the nasal chamber and sinuses and the superior orbital fissure. Although a routine decompression of the orbit is done in cases of exophthalmos, it relieves this sign only partially.

Dr. Trueman stated that the cause of the exophthalmos is probably venous engorgement. This explanation seems most likely, because the structures of the orbit are edematous and under tension, as though there was obstruction to the venous drainage system.

A plea is made for the early recognition of these tumors. As Dr. Trueman has stressed in his paper, when a patient comes with slight primary atrophy of the optic nerve, a temporal field defect in the same eye and roentgenographic evidence that the pituitary fossa is of normal or upper normal size, he should be studied without delay for the possibility of a tumor of the lesser wing of the sphenoid bone.

DR. WALTER I. LILLIE: I should like to go one step further in discussing the early stage of the ocular syndrome.

An early sign is the retrobulbar neuritis syndrome on the side of the lesion, that is, lowered visual acuity, a central scotoma and otherwise normal findings. This is the ideal time for the diagnosis to be made, as it permits the neurosurgeon to enter and remove the tumor before it has been of long enough duration to have produced atrophy of the optic nerve. Optic atrophy is irreversible, a fact which I wish to stress even more strongly than Dr. Groff did.

The ophthalmologist sees the patient first, and is the one responsible if there is delay in reaching the neurosurgeon. In the early stages the fundus is usually normal, and it is necessary to differentiate the lesion from an inflammatory or toxic condition. Against that diagnosis, as Dr. Trueman brought out and Dr. Groff has stressed, is the insidious onset, and slowly progressive character. These features speak for tumor rather than for inflammation or vascular lesion. I feel that in these cases a thorough neurologic examination is necessary, and should be requested by the ophthalmologist. I also strongly advocate making an encephalographic study early, for I believe that it is helpful to look for basal arachnoiditis in the sulcus chiasmatis, for lesions of the sphenoid ridge, calcification and a mass type of space-taking lesion. I concur with both Dr. Trueman and Dr. Groff, except I feel that it is necessary to detect the earliest ocular syndrome, and not wait for the appearance of optic nerve atrophy.

DR. BURTON CHANCE: I am reminded by this history of a case of my own. I had been consulted by a lawyer who was engaged in a suit concerning the death of a woman after the collision of the automobile in which she was riding with her family with a horse-drawn vehicle. Photographs made by bystanders confirmed the transportation company's contention that the company was not at fault; nevertheless, a verdict was entered against the company.

Before the injured woman's death, it was declared that her sight had been destroyed, and that she was blind. I succeeded in obtaining the record books of an ophthalmologist, then deceased, who, eleven years before the accident, had been consulted by the woman. He recorded that she was blind in her right eye, because of complete atrophy of the optic disk, which he regarded as resulting from the

high degree of progressive myopia in each eye. At that time there were no roentgenographic facilities, and further study was not made. In opposition to the plaintiff's family, an autopsy was obtained. Attached to the sphenoid bone was observed a tumor about the size of a large white grape, extending by erosion into the orbit. Sandlike changes were felt over the surface of the tumor. By some strange slight of hand, the tumor was spirited away and was never recovered.

DR. ROBERT H. TRUEMAN: Dr. Groff and Dr. Lillie have both emphasized, as I have tried, the importance of the early changes in the diagnosis of this tumor.

I should like to call attention to the fact that this analysis includes cases representing all sizes of these tumors, from the largest, in the early Cushing series, to the smallest, in the series of Groff and of Elsberg and Dyke. This variability in size explains the various types of clinical pictures, from the very early changes in the fundus and field to bilateral blindness. While it is important to recognize the very early changes, one should know that as the tumor grows progressive changes occur, for it is possible that a case might just as well be seen late as early.

I should like to ask Dr. Lillie a question. In his experience, has he ever been able to pick up a case of meningioma of the lesser wing of the sphenoid bone in which there was only a central scotoma?

DR. WALTER I. LILLIE: Yes, I have.

DR. ROBERT H. TRUEMAN: That answers my question. Thank you.

Protection and Maintenance of Night Vision for Military Personnel. DR. ROBERT H. PECKHAM.

This paper is a report of the recommendations and research performed by the Vision Committee of the National Defense Research Committee for the military services during the war. The author acted as liasion officer between the Committee and the Bureau of Medicine and Surgery.

As a result of the deliberations of this group of civilian and uniformed scientists, the following action was undertaken by both the Army and the Navy.

1. All personnel were instructed in the use of the eyes at night, with especial attention to waiting for dark adaptation and to using the rods of the retina by "looking around things."

2. An attempt was made to supply night combat personnel with red goggles, to preserve dark adaptation and to permit the attainment of adaptation in lighted compartments.

3. All Navy personnel were examined to eliminate night-blind persons.

4. As a result of directed research, it was decided that sunglasses were necessary for day wear, to permit best night vision. These sunglasses were neutral in color, not green or amber, of 10 per cent transmission and polarized, with the plane of transmission vertical. Finally, no specification against transmission in either ultraviolet or infra-red light was included.

5. The facts learned during the war can be applied to peacetime pursuits. Since the effect of sunlight is to reduce the sensitivity of the retina, sunglass protection is recommended for fluoroscopists. The effect of sunlight is to reduce the efficiency about 30 per cent. Sunglasses are needed by all persons engaged in colorimetry or any visual task involving comparative judgment. The effect of sunlight reduces retinal efficiency by from 20 to 50 per cent, depending on the task.

In the determination of avitaminosis by measuring the course of dark adaptation, the effect of previous exposure to sunlight could conceivably be so great as completely to confuse the readings. Exposure to sunlight can be effective as long as three weeks after the period of exposure; hence, the effect of vitamin therapy can be lost to demonstration.

All persons driving in dusk and at night after a day in unprotected sunlight will lose at least half their night visual efficiency, thereby rendering their driving dangerous, uncertain and fatiguing.

DISCUSSION

DR. BURTON CHANCE: In commenting on the statement that persons exposed for long hours in the glaring sunlight were found to have distinct loss of power at night, I have noticed on my repeated visits to tropical islands that my native friends seldom enjoyed walking out with me at night, because they declared they could not see well at night. Visitors from northern climates have been impressed by the suddenness of nightfall—it is as though the sun had suddenly dropped out of the heavens and darkness was instantaneous.

On long voyages, as once on a sailing ship, before I entered ophthalmology, I noted how sailors, who had been most capable during the daylight hours, became less efficient during the night, especially after the periods in which their daytime watches were prolonged.

In World War I, at my Army hospital, located at the seaside, men from northwestern climates, who delighted in spending all the daylight hours on the beach, preferred to remain indoors at night. I believe that the daylight glare interfered with the photochemical reactions in the retina.

DR. WALTER I. LILLIE: I should like to have a word before Dr. Peckham closes. One reads so often in the newspapers that the Negro race, the yellow race and the brown race have much better night vision than the white race. I wonder whether any of his investigations would prove or disprove such statements.

DR. ROBERT PECKHAM: My associates and I made many measurements designed to distinguish racial differences. The first were made by the Army for the purpose of selecting drivers of trucks and jeeps under black-out conditions. Members of the Negro race were deliberately chosen because it was suspected that, being highly pigmented, they would have deeper choroidal pigmentation and be better able to see at night. The tests showed no difference. Many more careful and extensive tests were made during the war, until it was finally concluded that no one race showed any superiority of retinal sensitivity.

Persons of some races were better able to perform out of doors at night, being better trained for night vision because of their savage

environment. At one time early in the investigations, we got a hurry-up call from the British that it had been suggested that the Japanese were night blind. Because they were myopic, something ought to be the matter with them. Fortunately, a few records of retinal sensitivity were available from Japanese students, and these showed no differences in retinal sensitivity.

During the Pacific campaign, the Marines reported hearing Japanese broadcasts to the effect that the American Marines should stay indoors at night. It was purported that certain selected Shinto fighters had been chosen for operations on the retina including the injection of secret drugs, and were then trained to be night killers. These persons, it was boasted, had such tremendous retinal sensitivity that they could not go out of doors in the daytime, and therefore would not be seen. This sounded like a fairy tale, but it had to be investigated. It was concluded that it was a falsehood, invented by the Japanese because they had discovered that they had actually lost their night retinal sensitivity. They had occupied the islands for months, and even years, waiting and watching for us to come. We knew they were aware of their loss of night vision, for we found their foxholes literally lined with boxes of vitamin A concentrates. We had ourselves tried such concentrates and found that they were useless to replace lost retinal sensitivity. Only time, and considerable time at that, will permit the retina to regain its lost sensitivity.

Book Reviews

Proceedings of the All-India Ophthalmological Society. Vol. VIII. Pp. 176. Madras Publishing House, Ltd., Mount Rd., Madras, 1945.

The eighth meeting of the All-India Ophthalmological Society was held at Hyderabad, Deccan, on March 8 to 10, 1945, under the presidency of Dr. E. V. Srinivasan, of Madras.

The president, in his introductory address, listed transplantation of the cornea, gonioscopy and penicillin therapy under the head of recent advances. Twenty-four communications, on a great variety of clinical subjects, were presented. Glaucoma occupied the principal place and was discussed under the headings of etiology, symptomatology, treatment, Bengal epidemic dropsy glaucoma, secondary glaucoma and glaucoma and gonioscopy.

ARNOLD KNAPP.

Transactions of the Ophthalmological Society of Australia (British Medical Association). Volume 4. Pp. 212. Price, 7s.6d. Sydney, Australia: Australasian Medical Publishing Company, Ltd., 1944.

This is a report of the proceedings of the Ophthalmological Society of Australia in the first meeting held in three years, on Oct. 12, 1944, in Melbourne. The presidential address was delivered by Dr. G. B. Black, who succeeded to office on the death of the president, Dr. J. L. Gibson.

E. C. Black discussed ocular injuries in industry, reporting 10 cases of various injuries, with special reference to accidents occurring on railroads. Articles on lighting, particularly in industry, were read by L. D. Wright, who made a plea for medical leadership in this project, and by A. L. Tostevin, who called for a visual survey of school children.

Certain ocular disorders found in the tropics were listed in papers by J. A. Pockley, K. B. Armstrong and Clifford Colvin. The diseases described by the first two authors (among which were dendritic ulcer, quinine amblyopia, melanoma of the choroid and dacrycystitis) are not particularly confined to the tropics, although Pockley mentioned that scrub typhus may be accompanied with thrombotic changes in the retinal vessels, subconjunctival hemorrhage and corneal ulcer.

Colvin, however, confined his report to conditions indigenous to the tropics. He stated that he had encountered dendritic ulcer, hyperemia of the disks and retinal hemorrhages with malaria; painful ocular movements and sustained asthenopia with dengue; iritis with relapsing fever and dysentery, and corneal tumors with leprosy. Herpes simplex of the cornea occurring in patients with malaria was described by W. P. Chamberlain and L. H. Bronson, medical officers in the Army of the United States. They reported six times as many cases of such corneal involvement in a malarial division exposed to malaria as in a division not so exposed. They recommended local application of sulfadiazine powder to the lesion.

Lessons to be learned by ophthalmologists from the war were summarized in a thoughtful paper by J. B. Hamilton. His suggestions included utilization of opticians in private practice, an improved supply of ophthalmic nurses, an increase in special hospitals for ocular diseases,

the establishment of private ophthalmic clinics in the larger cities, ophthalmic training for discharged medical officers and the stimulation of laboratory and research work by the society. Hamilton also read a paper on tuberculous lesions of the eye. His classification follows.

1. Undoubted infection of the globe with tubercle bacilli
   (1) Uveitis with mutton fat keratic precipitates and Koeppe nodules
   (2) Uveoparotid fever
   (3) Conglomerate tuberculosis of the retina
   (4) Miliary tubercles on the retina associated with fulminating miliary tuberculous meningitis
2. Tuberculous lesions of the globe in immune desensitized persons
   (1) Heterochromic cyclitis with or without complicated cataract
   (2) Disseminated choroiditis with loss of pigment
   (3) Retinal periphlebitis with hemorrhage in the vitreous and retinitis proliferans—Eales' disease.
3. Tuberculous lesions of the globe in immune sensitized persons. Phlyctenulosis
4. Doubtful tuberculous lesions of the globe
   (1) Interstitial keratitis
   (2) Sclerosing keratitis
   (3) Episcleritis and scleritis
5. Pseudotuberculous lesions, Boeck's sarcoid

He stressed the value of a history of contact and urged removal of the patient from his or her source of infection.

There were four papers on congenital cataract associated with rubella in the mother. Ida Mann, summarizing the observations already made (which, incidentally, originated chiefly in Australia), pointed out that the period of susceptibility of the lens is between the first and the second month. She wisely stated:

> It has never been the custom for physicians to make searching inquiries concerning illnesses occurring at the very beginning of pregnancy. This, undoubtedly, should be done in every case of congenital abnormality, when it may well be found that a variety of causes other than rubella operating during the first six weeks may produce congenital cataract and heart disease. If such congenital disorders should be found specific for rubella, it would be surprising from the embryologic point of view. Meanwhile, the observations stand for an important advance in the correlation of experimental and clinical findings.

N. McA. Gregg added to his previous contribution (Congenital Cataract Following German Measles in the Mother, *Tr. Ophth. Soc. Australia* **3**: 35, 1941) and listed certain problems still to be solved —isolation of the virus of rubella; development of prophylactic and curative treatment; more publicity in order to prevent infection of the potential mother, and care and treatment of the defective child resulting from the disease. Similar observations and recommendations were given by Charles Swan. F. V. Scholes, medical superintendent of the Hospital of Infectious Diseases, brought up the interesting point that other virus diseases, notably measles, may, if sufficiently severe, produce similar fetal malformations.

In a paper on "Crossed Cerebral Dominance" (in which the master hand and the master eye are on opposite sides), J. R. Anderson and

C. Weigall made this difficult and important subject clearly understandable to the ophthalmologist. The whole article should be read, but their conclusions are as follows:

1. Most children demonstrate naturally a preference for one or the other hand. This preference appears to be inherited. It requires practice before it becomes fixed. The strength of its fixation varies greatly with the individual. In some persons transference of mastery to the other hand is difficult, while in others it can occur without upset.

2. The dominance of one eye is probably also inherited.

3. The occurrence of crossed dominance is likely to be associated with nervous symptoms. In addition, difficulty in learning to fly, in playing games, and even in routine school work, is sometimes experienced. These, and crossed dominance, are more probably concomitant signs of an imperfectly integrated nervous system than the results of crossed dominance.

4. Ocular dominance may affect the prescription of lenses if both eyes are open while the nondominant eye is being tested.

5. Difficulty in developing fusion is the rule in persons with convergent squint if crossed dominance is present.

6. It appears that if crossed dominance is always an obstacle its influence may be obscured by special development of such qualities as determination, enthusiasm and physical fitness.

7. Not only physical but emotional and intellectual gains may follow the careful unilateralization of ocular and manual dominance in selected persons.

Other, and shorter, articles in the volume were those by A. Joyce, who reported a case of glioma of the retina cured by the implantation of a radon seed; by N. M. Macindoe, who advocated the cure of dacryostenosis by inserting a small rubber tube in the lower end of the lacrimal sac and placing it in an opening into the nose (the tube was subsequently removed); by J. L. R. Carter, who described his procedure—apparently the universal one—of probing for infantile dacryostitis; by K. O'Day, who wrote somewhat inconclusively on the color of the human iris; by E. V. W. Pockley, who stated the advantages of intracapsular cataract extraction; by A. d'Ombrain, who stated that he prefers making his cataract incision with a keratome, and by L. Gilchrist, who recommended partial occlusion in the treatment of amblyopia ex anopsia.

An article on repair of the eyelids and periorbital structures was contributed by B. K. Rank, who apparently is a plastic surgeon rather than an ophthalmologist.

Taken as a whole, this volume can be read with pleasure and profit and clearly indicates that Australian ophthalmology is in the first rank.

G. M. Bruce.

Transactions of the Ophthalmological Society of Australia (British Medical Association). Volume 5. Price, 7s: 6d. Pp. 143. Sydney, Australia, Australasian Medical Publishing Company, Ltd., 1946.

In this volume are reported the proceedings of the fifth general meeting of the Ophthalmological Society of Australia, held in 1945. The opening address was delivered by the president, N. McAlister Gregg.

In a lengthy, but well arranged, paper (which takes up about one third of the volume) C. G. McDonald discusses diseases of the arteries and their relation to the eye. He suggests that the word "arteriosclerosis" should be abandoned, as it is too general a term. He believes that when the subintimal layer of an artery is diseased "atheroma" is the term to be employed. This condition is rarely detected in retinal vessels but may coexist with "essential hypertension" (hypertensive vascular disease). The latter is the dominant disease of arteries. McDonald divides this disease into three stages: (1) functional vascular hypertonus; (2) vascular and visceral reactions—cardiac hypertrophy, medial hypertrophy of small vessels and diffuse hyperplastic sclerosis of visceral vessels, and endarteritis obliterans—and (3) visceral failure—cardiac defeat, cerebral vascular accidents and renal failure. "Malignant hypertension" (rapidly progressive hypertensive vascular disease) he discusses separately, although he is not convinced that it is a separate entity from hypertensive vascular disease. In fact, he believes that all cases of hypertensive vascular disease will become rapidly progressive if the diastolic pressure rises high enough. Nephritis he classifies as (1) acute focal glomerulonephritis, due to a primary infection elsewhere, usually in the throat; (2) acute focal embolic glomerulonephritis, occurring in the course of subacute infective endocarditis; (3) acute diffuse glomerulonephritis, also due to a focal infection but later in onset; (4) subacute diffuse glomerulonephritis, with gross albuminuria but without elevation of blood pressure, and (5) chronic diffuse glomerulonephritis, the familiar chronic stage.

In describing the fundus oculi in hypertension and nephritis, he contributes little that is not already known to ophthalmologists. He believes that no differentiation can be made ophthalmoscopically between the retinal picture of hypertension and that of nephritis. However, he also states that diabetic retinitis cannot be diagnosed with the ophthalmoscope.

P. L. Spero discusses some ocular manifestations due to malnutrition as seen in returned prisoners of war recently released by the Japanese. He examined 64 patients. He states that the most frequently encountered ocular lesion was retrobulbar neuritis followed by partial atrophy of the optic nerve affecting the papillomacular bundle. His observations are confirmed and added to by F. P. Claffy, who had himself been a prisoner of war for three years. He had discovered that retrobulbar neuritis and corneal degeneration followed deprivation of vitamin B complex, and partial ophthalmoplegia resulted from the absence of thiamine. He found impairment of vision (from 6/5 to counting of fingers); fine nystagmoid movements, occasional mild papillitis, going on to temporal pallor; rare retinal hemorrhages; paracentral or central scotomas (relative or absolute), and normal peripheral fields. The prognosis depended on the amount of atrophy that had supervened. While frank corneal ulceration occasionally was encountered, more frequently a noninflammatory diffuse superficial degeneration occurred. Permanent opacification was rare.

Darcy Williams speaks of the fitting of contact glasses, describing a technic which is similar to that used in the United States.

Three cases of toxoplasmic encephalomyelitis and chorioretinitis are reported by E. G. Robertson and J. B. Hamilton. All the patients

were girls, aged 13, 5 and 16 years, respectively. One had convulsions; 1 was of below normal intelligence, and the other, an inmate of an institution, was unable to read or write. All had choroidal changes, and calcified deposits were demonstrated in the roentgenograms.

Granville Waddy reports a new ocular syndrome, consisting of poor vision (6/60 to 6/36), nystagmus, albinoid fundi and a high degree of intelligence. He had 8 or 10 patients with this syndrome.

There are papers on ocular sarcoidosis by H. Ryan and W. D. Counsell and by the latter author with J. B. Hamilton. In all, 3 cases are reported. Conservative treatment is advocated, as the outlook is good. Every effort should be made to distinguish Boeck's sarcoid from tuberculosis.

In discussing the treatment of secondary cataract, E. T. Smith states that early iridectomy is indicated. If extraction is too long delayed, the eye may degenerate. An interesting feature of this paper is an account of the ocular vicissitudes of James Joyce, whose eyes were operated on more than seven times for cataract secondary to iritis.

F. G. Roberts' patients had distressing psychologic reactions following cataract extractions, and he advocated obtaining a state of complete "non-cooperation" in every case. In a paper entitled "Premedication and Cooperation in Cataract Extraction," he suggests giving the patients sodium amytal and eliminating the movements of the eyeball by injection of procaine. The eye could be turned downward by stitches inserted into the superior rectus or on each side of the cornea. In the ensuing discussion, there was evident a courteous lack of enthusiasm over this technic.

Prolapse of the iris, according to N. M. McIndoe, is less likely to occur after a simple intracapsular extraction followed by suturing of the wound, instillation of physostigmine and ample postoperative sedation. The prolapsed area should be immediately incised and replaced. Cautery is contraindicated.

Arthur D'Ombrain believes that traumatic monocular glaucoma is a clinical entity. He lists 18 cases, in all of which three common factors were present: (1) a history of trauma, (2) glaucoma in one eye and (3) absence of glaucoma in the other eye. He suggests that some types of concussion shock can upset the stabilization of intraocular pressure.

C. H. B. Black reports 3 cases of keratitis which he believes were due to deficiency of ascorbic acid. He asks for further clinical investigation of the subject by others.

A case of septic thrombosis of the cavernous sinus is reported by R. G. Banks-Smith. After severe headache and delirium, double proptosis appeared, and there was venous engorgement in one fundus, the other being invisible because of edema. The patient recovered under penicillin and sulfonamide therapy, with normal vision in one eye but with atrophy of the optic nerve and blindness in the other.

The volume ends with an obituary of the late Sir James Barrett. This olympian figure, one of the great men of the Empire, had begun his career as an ophthalmologist.

G. M. Bruce.